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## CALCULATING BIFOCAL IMBALANCES

This is the first of a series of seven articles on calculating and correcting vertical imbalances in bifocal segments.

Whenever there is a marked difference in the corrections required for the two eyes, it is well to suspect an induced vertical imbalance. The method of calculating this imbalance is as follows: At a point 8 mm. below the optical center of the distance lens, the prismatic effect is calculated by multiplying the distance correction by .8. Thus, a +1.00 lens at this visual point 8 mm. below the distance center would produce .8 of a prism diopter. Since the lens is plus, the prism would be base up. If the lens were minus, the prism would be base down.

Let us take a correction that reads: Right eye +1.00, Left eye —1.00. This combination of corrections would produce a vertical imbalance of 1.6, because the left eye produces .8 base up and the right eye .8 base down, the resulting imbalance being 1.6.

Again, if the right eye required +1.00 and the left eye +2.00, there would be an imbalance of .8, due to the fact that at the visual point the right lens produces .8 base up and the left lens produces 1.6 base up, the difference being the amount of induced imbalance.

It will be seen that when one eye requires plus and the other eye requires minus, the total of the imbalance is found by adding the two prism values. When both are plus or minus, the imbalance is found by subtracting the lesser from the greater.

So far we have talked only about spherical corrections. If, and there generally is, a cylinder to be calculated, a special chart is required. This chart will be published in the April issue of the "Scientific Corner," followed by an explanation in the June issue. The "Scientific Corner" will appear every other month until this series is completed.

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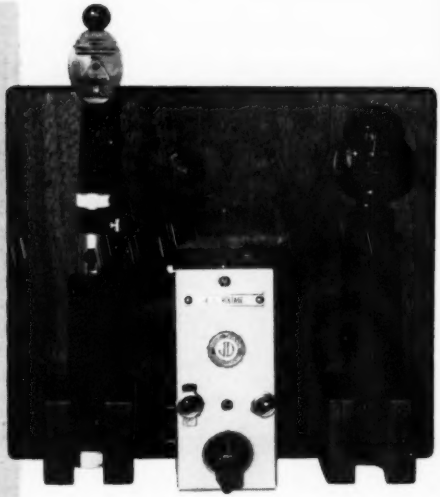

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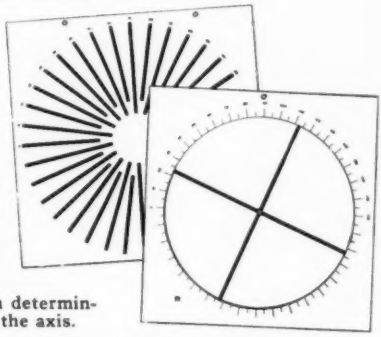

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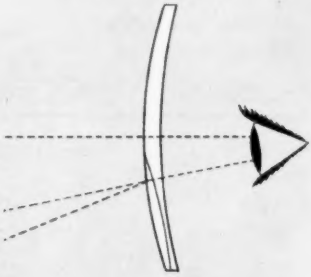

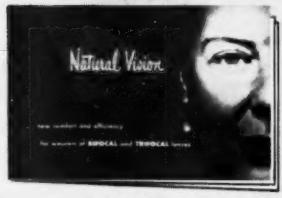


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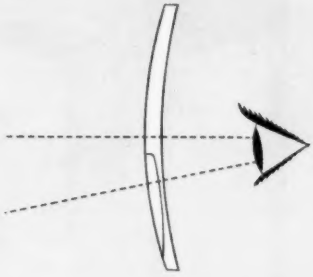

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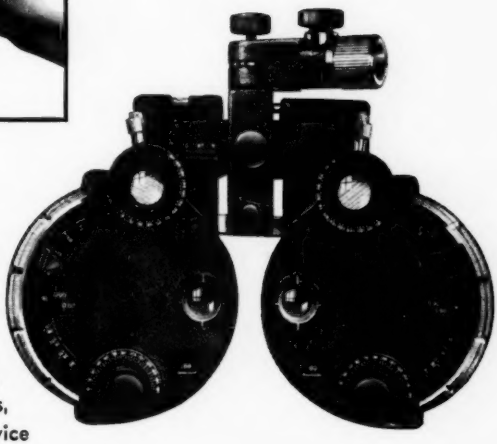

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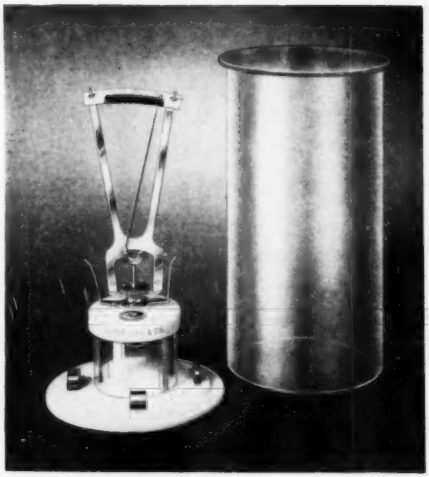

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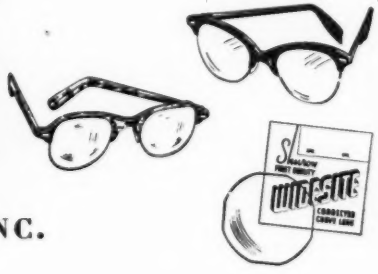


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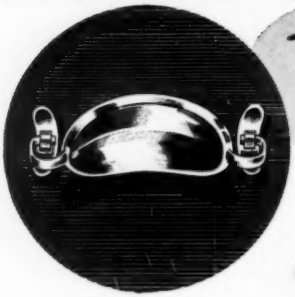

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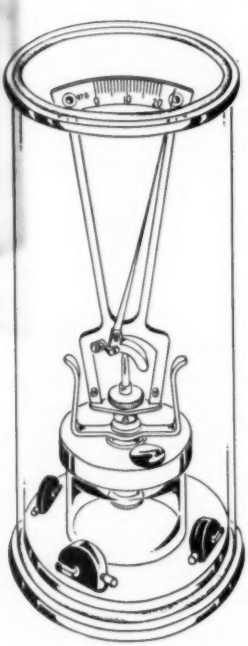

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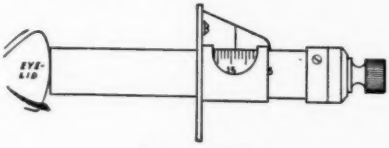


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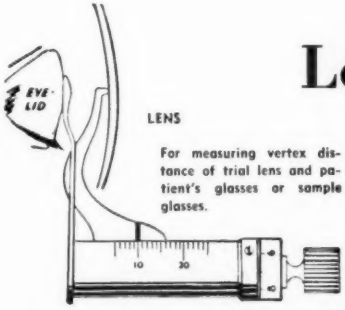


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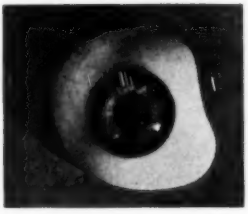

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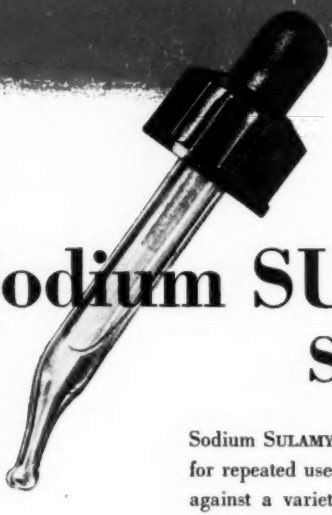

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# DISTORTION OF THE IMAGE BY OPHTHALMIC PRISMS

KENNETH N. OGLE, Ph.D.

ROCHESTER, MINN.

THAT THE image seen through an ophthalmic prism is distorted is well known to every refractionist, and this distortion is especially apparent through large prisms, and even through the usual Risley rotary prisms. Although known qualitatively, a general quantitative discussion of this distortion has never appeared in the literature. This situation can be accounted for not only because of the mathematical complications involved but also, and probably more so, because of the general lack of interest in such distortion, except as a phenomenon to be tolerated. In 1927 Hartinger [1] calculated the magnitude of this distortion for particular prisms with plane surfaces, especially those of high deviating power. He did not give a quantitative description that was applicable to ophthalmic prisms in general.

There has been more interest of late in this distortion because of its influence on stereoscopic spatial localization when ophthalmic prisms are worn before the two eyes.[2] The quantitative description of the distortion [3] and the problem of changing the magnitude of the distortion by grinding the prisms on different base curves become more important. Of course, the apparent distortion of stereoscopic spatial localization caused by ophthalmic prisms has been known for a long time; for example, Nagel [4] was aware of the phenomenon. To such distortion has been attributed the inability of some persons to wear prismatic corrections.

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From the Section of Biophysics and Biophysical Research, Mayo Foundation, the University of Minnesota, and the Mayo Clinic.

1. Hartinger, H.: Die Verzeichnungsfehler einfacher Prismen, Ztschr. ophth. Optik. **15-17**:129-146, 1928-1929.

2. (*a*) Ames, Adelbert, Jr.: Binocular Vision as Affected by Relations Between Uniocular Stimulus-Patterns in Commonplace Environments, Am. J. Psychol. **59**:333-357, 1946. (*b*) Ogle, K. N.: Researches in Binocular Vision, Philadelphia, W. B. Saunders Company, 1950. (*c*) Lancaster, W. B.: Some Secondary Subjective Effects Produced by Prisms, Tr. Am. Ophth. Soc. **46**:262-283, 1948.

3. Miles, P. W.: Eliminating Distortion Due to Prisms in Glasses, Am. J. Ophth. **34**:87-93, 1951.

4. Nagel, A.: Die Anomalieen der Refraction und Accommodation des Auges: in von Graefe, A., and Saemisch, T.: Handbuch der gesamten Augenheilkunde, Ed. 1, Leipzig, W. Engelmann, 1880, Vol. 6, Chap. 10, p. 366.

The apparent distortion of the image through an ophthalmic prism depends not only on the physical dimensions of the prism but also on its distance from the eye and its orientation before the eye. In effect, the phenomenon is evidence of a different prismatic deviation for rays that reach the eye and traverse different parts of the prism. Since the diameter of the cone of light from a point in space is limited by the size of the pupil of the eye, only a small part of the prism is traversed by that cone. That cone which arrives at the eye from a point to the left will traverse the prism in an entirely different part than that from a point to the right. In the two instances, the angles of incidence and the lengths of paths of the rays through the glass will be different.

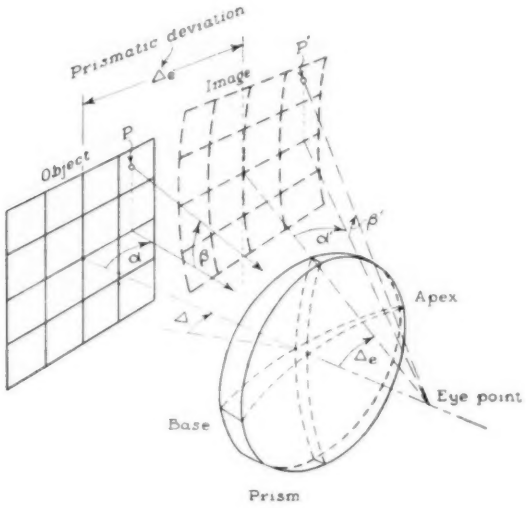


Fig. 1.—The image pattern depicted in this drawing must not be taken literally but should be understood to indicate only a directional pattern, not a specific spatially located image. This "directional" image pattern will change with distance and orientation of the prism before the eye. The object pattern will be considered to be at a great distance from the eye.

Figure 1 illustrates schematically for an ophthalmic prism the pattern of directions of the image rays relative to a given eye point, corresponding to a given object pattern. The distortion is characterized by the following factors: 1. A changing angular magnification of the image in the base-apex meridian (described here as the horizontal meridian) with lateral angle. This is usually an increase in magnification toward the apex of the prism. Thus images appear progressively stretched in the base-apex meridian toward the apex. 2. A curvature of the images of vertical lines, that is, those lines perpendicular to the base-apex meridian, or parallel to the base. The bulge of the curvature is always toward the base of the prism. 3. A change in the angular magnification in the vertical meridian with lateral angle. This results in an apparent slanting of all lines parallel to the base-

apex meridian and a wedge-shaped appearance of parallel horizontal lines. There appears to be no other distortion in the vertical meridian,[3] at least out to visual angles of 20 degrees. In addition to these specific distortions, there will be a general angular magnification of the image, which may be slightly different in the horizontal (base-apex) meridian and in the vertical meridian.

Ophthalmic prisms in general are ground with spherical (and even toric) surfaces, with or without refracting power. Only trial-case prisms have plane surfaces. The distortion[4] of the ophthalmic prism will vary with (1) the prism power, (2) the surface curves (base curves), (3) the distance of the prism from the eye, (4) the thickness of the prism, (5) the refractive power, and (6) the orientation of the prism before the eye.

Since the magnitude of the distortion varies with the distance of the prism from the eye, the magnitude of the distortion of the dioptric image on the retina

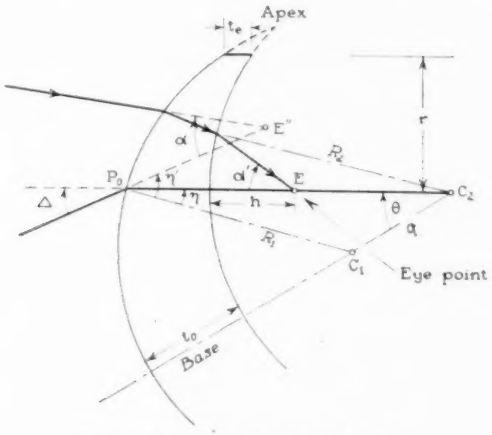


Fig. 2.—The principal section of a prism.

will differ slightly from that of the distortion which will influence the fixation movements of the eye behind the prism. This follows because the magnification of the image on the retina will be determined by the position of the entrance pupil (we are dealing essentially with blurred imagery [2b]), whereas the magnification of ocular excursions of the eye will be determined by the position of the center of rotation of the eye. These points differ in position, behind the prism, by nearly 10 mm. Only in the case of flat prisms is the distortion independent of the distance of the prism from the eye. The angular magnification, and thus the distortion, will be specified in this report relative to an eye point, whether that point be chosen as the entrance pupil or as the center of rotation of the eye.

The geometric relations of an ophthalmic prism in the base-apex-eye meridian are shown in Figure 2. Such a prism can be specified by the two radii of curvature of the front and back surfaces, $R_1$ and $R_2$, respectively; the axial thickness, $t_0$, and

the angle $\theta$ between the line determined by the centers of curvature and the chief ray that passes through the eye point and the center of curvature of the back surface. For the axial-thickness specification, we may use the apex-edge thickness if we specify the radius of the lens. The primary prismatic deviation will be the angle $\Delta$. A distant object point which subtends an angle $\alpha$ will be imaged to give the angle $\alpha'$. Thus, only 4 parameters are needed to specify the prism, 1 more than the number needed to specify the dimensions of an ophthalmic lens.

Now it has been shown [5] that the angular dimensions of the image seen through an ophthalmic prism for angles within about 20 degrees can be adequately described by the two equations.

$$\begin{aligned} \alpha' &= A\alpha + B\alpha^2 + C\beta^2 \\ \beta' &= D\beta + E\alpha\beta \end{aligned} \qquad (I)$$

In these equations, $\alpha$, $\beta$, and $\alpha'$, $\beta'$, are the directional azimuth (lateral) and elevation (vertical) angles (cf. Fig. 1) of the object and image patterns, respectively, corrected for the prismatic deviation. The coefficients that occur in these equations are as follows:

**A**, the angular magnification in the horizontal, or base-apex, meridian, which depends essentially on the power of the lens, the curvature of the front surface, and the thickness of the lens.

**B**, the magnitude of the unsymmetric angular magnification in the base-apex meridian.

**C**, the term that describes the effective curvature of the images of vertical lines. This description implies that these images are parts of parabolas.

**D**, the angular magnification in the vertical (parallel to the base) meridian. This angular magnification depends on the same factors as does **A**. That **D** may differ slightly from **A** probably results from the fact that most rays passing through the prism are actually rays at oblique incidence and hence become slightly astigmatic.

**E**, the magnitude of the change in vertical magnification with lateral angle. This is also the slope (slant) of the images of a horizontal line at a vertical angle $\beta$ above or below the base-apex-eye meridian.

The analytic problem was to express the coefficients **A** to **E**, inclusive, in terms of the physical dimensions of the prism and the distance of the prism from the eye point. These, of necessity, appear complicated, because there are so many variables involved, namely, base curve, $R_1$ (or $R_2$), the radii of curvature of the front and back curves; vertex power, $l'_0$; prismatic deviation, $\Delta$; thickness of prism or apex-edge thickness, $t_a$, for a lens $2r$ in diameter, and the distance, $h$, of prism from the eye point. The prism is assumed to be oriented as is usual, with the back surface normal to the primary direction of the object. It has been shown, however, that

5. Ogle, K. N.: Distortion of the Image by Prisms, J. Optic Soc. America, to be published.

$$A = 1/a(1 + NQ) \qquad B = \tfrac{1}{2}a^2A(N^2P - Q)$$

$$C = \tfrac{1}{2}aD\left[ND - TA\right] \qquad D = 1/aS$$

$$E = aD\left[TD - NA\right]$$

and in these

$$a = \left[R_2 - (n-1)h\right]/nR_2$$

$$N = \cot\theta + \left[R_2(R_2 + h)\right]/R_1\left[R_2 - (n-1)h\right]k$$

$$k = \sin\triangle\left[1 + n^2 - 2n\cos\triangle\right]^{-\frac{1}{2}} = \sin\eta$$

$$S = \cos\triangle + N\sin\triangle$$

$$T = N\cos\triangle - \sin\triangle$$

$$\cot\theta = \left[(R_2^2 - r^2)^{\frac{1}{2}} + t_e - R_1(1 - L^2)^{\frac{1}{2}}\right]/R_1k$$

$$L = r/R_1 + k$$

$$V_0 = D_1/(1 - D_1t/n) + D_2;\ D_1 = (n-1)/R_1;\ D_2 = (n-1)/R_2$$

$$t = R_1(1 - k^2)^{\frac{1}{2}} + R_1k\cot\theta - R_2$$

$n$ = index of refraction

$$Q = \tan\eta' - \tan\eta\ ;\ \eta' - \eta = \triangle$$

$$P = \tan^3\eta' - \tan^3\eta$$

$$\sin\eta' = n\sin\eta$$

The various formulas involved appear to be very complicated. Actually, they are relatively simple expressions. Given the dimensions of a prism, the coefficients can be readily calculated and the distortion then described. The advantage of this description is that we can discuss any one of the aspects of the distortion and the factors on which that aspect depends. Another advantage of these coefficients is that they allow us to determine by calculation what the dimensions of a prism must be in order to give certain types and magnitudes of distortion, or no distortion.

Actually, of course, the eye turns behind the prism through the angle $\triangle_e = \mathbf{A}\triangle + \mathbf{B}\triangle^2$. Then equation 1 becomes

$$\alpha' = (A + 2B\triangle)\alpha + B\alpha^2 + C\beta^2$$
$$\beta' = (D + E\triangle)\beta + E\alpha\beta \qquad (2)$$

In Table 1 is shown the comparison of calculated and measured [5] values for the several coefficients of distortion for a 5△ afocal prism ground on a 6.25 D. base curve, with a diameter of 42 mm. and apex-edge thickness of 1.2 mm. This prism was

placed a vertex distance of 15 mm. from the eye point. These measurements were obtained on the special testing instrument previously described.[6]

These general formulas hold also for flat prisms—those with plane surfaces, that is, when $R_1$ and $R_2$ are infinitely large. The coefficients reduce to:

$$A = (n \cos \triangle - 1)/(n - \cos \triangle); \quad B = \tfrac{1}{2}A(n^2 - A^2)(n \sin \triangle)/n^2(n \cos \triangle - 1);$$

$$C = \tfrac{1}{2}(n^2 - 1)\sin \triangle / n(n - \cos \triangle); \quad D = 1, \quad E = 0$$

For a flat prism, then, the various aspects of the distortion depend only on the prism deviation, $\triangle$, or the prism apex angle, and the index of refraction; they are independent of the distance of the prism from the eye and of the thickness of the prism. The angular magnification in the vertical meridian (parallel to the base) is unity (**D**=1); furthermore, this magnification does not change with lateral angle (because **E** is identically zero).

The dependence of the unsymmetric angular magnification, **B**, on the surface powers of the front surfaces of afocal prisms of three prismatic deviations is illus-

TABLE 1.—*Comparative Calculated and Measured Values for an Afocal 5△ Prism Ground with a Back-Surface Power of 6.25 D.*

| Coefficient | Computed Value | Measured Value |
|---|---|---|
| **A** (Angular magnification in base-apex meridian) | 1.0122 (1.22%) | 1.0129 (1.29%) |
| **B** * (Unsymmetric angular magnification in base-apex meridian) | 0.000349 | 0.000355 (change in magnification of 0.03% per degree of azimuth angle) |
| **C** * (Coefficient of curvature of images of vertical lines) | 0.00018 | 0.00016 |
| **D** (Angular magnification in vertical meridian) | 1.0157 (1.57%) | 1.0160 (1.60%) |
| **E** * (Change in vertical angular magnification with azimuth angle) | —0.000626 | —0.000614 (change in magnification of 0.06% per degree of azimuth angle) |

* Since these values pertain to angles $\alpha$ and $\beta$, expressed in degrees, the coefficients of column 2 are multiplied by $\pi/180$.

trated in Figure 3. The vertex distance from the eye point for the data in this graph is taken as 15 mm. The abscissas are the front surface powers, expressed in diopters; the ordinates are the values of **B**, expressed in per cent magnification change per 1 degree of lateral angle. The data to the right of the graph indicate that the prisms are placed with surfaces concave toward the eye, and those to the left, that the prisms are placed with surfaces convex toward the eye. The latter method of placement is rarely used except to introduce deliberately an unsymmetric magnification.[7]

The unsymmetric magnification is nearly directly proportional to the prism power and decreases with increase in power of the surfaces. However, this is nearly zero for the same surface power regardless of prism power. This surface power for which the unsymmetric magnification is zero varies markedly with the distance of prism from the eye point.

6. Ogle, K. N. and Ames, A., Jr.: Ophthalmic Lens Testing Instrument, J. Optic Soc. America **33**:137-142, 1943.

7. Ames, A., Jr.; Ogle, K. N., and Gliddon, G. H.: Lenses for Changing Size and Shape of Dioptric Images, Ann. Distinguished Serv. Found. Optom. **1**:61-70, 1932.

If we add dioptric power to the prism, this aspect of the distortion is also modified, as shown in Figure 4, where for a distance of 20 mm. the comparison is shown of +5 D. and —5 D. prism lenses, all having a 5△ deviating power. Here the

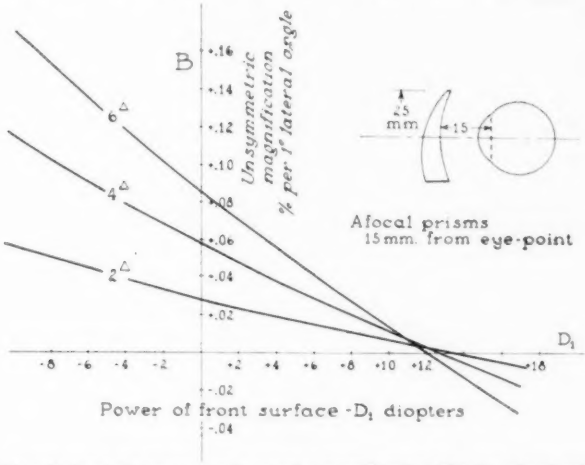


Fig. 3.—Magnitude of the unsymmetric angular magnification (**B**) for afocal prisms of various prism powers for a distance of the eye to the lens of 15 mm., which have different flexures, as defined by the surface power of the front surface.

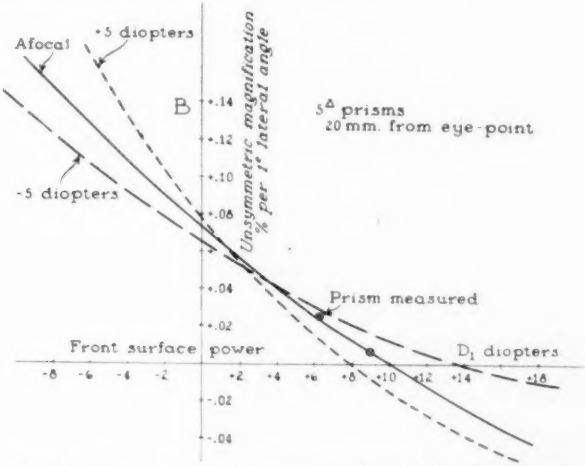


Fig. 4.—Change in the unsymmetric angular magnification (**B**) with change in front-surface power, for 5△ prisms at a distance of 20 mm. from the eye point, and for a plus or minus 5 D. power incorporated in the prism.

surface power at which this unsymmetric magnification is zero is very different. The spread between the curves increases for increasing distance of prism to eye, there being only a small separation between curves for distances less than 10 mm.

TABLE 2.—*Surface Powers for Ophthalmic Prisms* Necessary to Eliminate the Unsymmetric Angular Magnification Aspect of the Distortion*

| Vertex Distance, Mm. | | Vertex Power, Diopters | | | | |
|---|---|---|---|---|---|---|
| | | −5 | −2.5 | Afocal | +2.5 | +5 |
| 10 | $D_1$ † | ..... | ..... | 16.40 | 14.55 | 13.30 |
| | $D_2$ | ..... | ..... | −16.20 | −12.95 | − 9.25 |
| 15 | $D_1$ | ..... | 14.45 | 12.40 | 11.15 | 10.20 |
| | $D_2$ | ..... | −17.20 | −12.80 | − 9.10 | − 5.70 |
| 20 | $D_1$ | 13.70 | 11.10 | 9.80 | 8.90 | 8.10 |
| | $D_2$ | −18.55 | −13.80 | −10.05 | − 6.70 | − 3.45 |
| 25 | $D_1$ | 10.60 | 9.10 | 8.10 | 7.30 | 6.65 |
| | $D_2$ | −15.55 | −11.75 | − 8.30 | − 5.00 | − 1.85 |
| 30 | $D_1$ | 8.90 | 7.75 | 6.90 | 6.20 | 5.55 |
| | $D_2$ | −13.90 | −10.35 | − 7.00 | − 3.85 | − 0.90 |

* Prism power, 5Δ; diameter of prism, 50 mm.; apex-edge thickness, 1.0 mm.
† $D_1$ represents power of front surface, in diopters; $D_2$, power of back surface, in diopters.

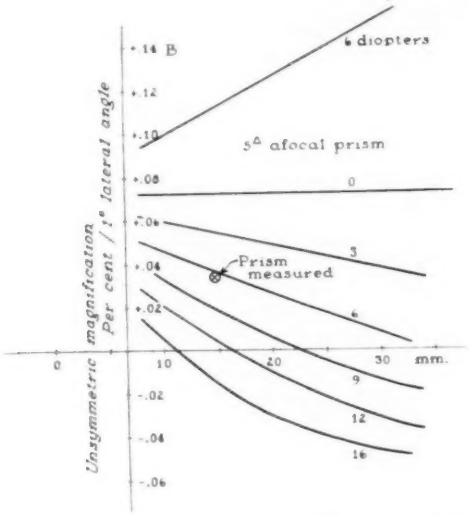


Fig. 5.—Rate of change of unsymmetric angular magnification with distance of prism from the eye for prisms ground on different base curves. These curves are for an afocal 5Δ prism.

We can determine uniquely [7] those surface powers for those prism lenses for which the unsymmetric magnification is zero by setting the coefficient **B**=0. Then $N=(Q/P)^{1/2}$. Representative of these surface powers for prism lenses, and for different distances between prism and eye point, are the calculated values given in Table 2.[8] An inspection of this table shows how sensitive is this aspect of the distortion to changes in the distance of the prism from the eye. It would appear, also, that it is the front surface which tends to be the important one for the determination of the distortion. The rate of change in this distortion with distance of prism to the eye point can be visualized also from Figure 5, for a 5Δ afocal prism.

8. These specifications, which agree with the actual measurements on ophthalmic prisms,[5] differ markedly from those given by Miles.[3] I am at a loss to understand how Dr. Miles arrived at his results.

The curvature of the images of lines parallel to the base of the prism cannot be eliminated by change of base curve. The curvature is always toward the base of the prism and increases with prism power, with distance of prism from the eye, except for the planoprism, and with negative lens power, but decreases with increase in curvature of the front surface of the prism. Figure 6 illustrates the magnitude (**C**) for this curvature, with change in base curve of the prism and in vertex power, for a 5△ prism. The data measured on a 4.7 D. prism on a 6.25 D. base curve are also shown. Inspection of the graph shows that the power of the prism lens affects the curvature of the images only slightly. No practical simple solution to the problem of eliminating this curvature is possible by change in base curve.

The remaining aspect of the distortion to be discussed is that of the change in the angular magnification in the vertical meridian (that parallel to the base of the prism) with lateral angle. This quantity is given by the coefficient **E**. Figure 7

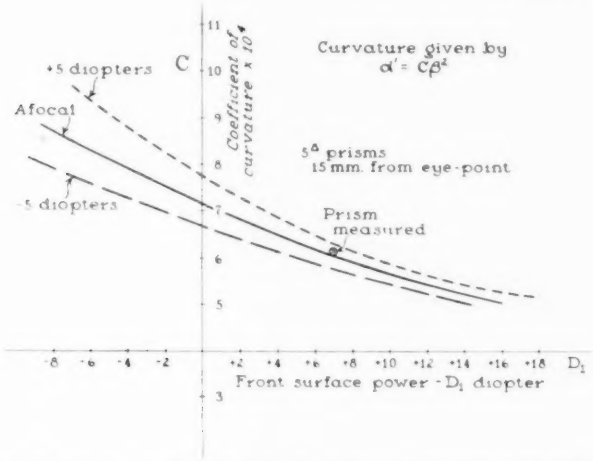


Fig. 6.—Magnitude of the coefficient of curvature of images parallel to the base of the prism and its change with change of base curve and with vertex power of the prism lens. These data are for a 5△ prism placed 15 mm. from the eye point.

illustrates the magnitude of **E** (in per cent magnification change for 1 degree of lateral angle) with powers of the base curve for afocal prisms and for plus and minus diopter powers. It will be noted that **E** is negative for prisms as usually placed before the eye (concave surface toward the eye). This means that the images of lines above the base-apex plane appear to slope up toward the base, and that those below appear to slope down toward the base. Only when the prisms are placed with convex surface toward the eye is **E** positive. It is important to note also that **E**=0 only when the front surface is a plane ($D_1$=0), this irrespective of the power of the prism lens or the distance of the prism from the eye. Thus, this aspect of the distortion of a prism can be eliminated by making the curve of the front surface flat.

The change in vertical angular magnification with lateral angle **E** increases (negatively) with the increase in distance of prism from the eye and with the

increase in prism deviating power and is greater for hyperopic corrections than for myopic corrections.

In addition to these three aspects of the distortion of the image seen through a prism, we must not overlook the regular angular magnification of that image in both the vertical and the horizontal meridian. This angular magnification is what is to be expected [2b] from the curvature of the front surface, the thickness, the vertex power, and the distance of the prism from the eye. The difference between the magnification in the base-apex meridian and that in the vertical meridian usually is of the order of a few tenths per cent, that in the vertical being the larger, so that essentially the angular magnification of the prism can be considered an "over-all" magnification.

If the prism is rotated about a vertical axis, the quantities discussed here are slightly changed, especially the prismatic deviation $\Delta_e$ [9] and the magnifications **A** and **D**. Much less affected are the distortion factors **B**, **C**, and **E**.

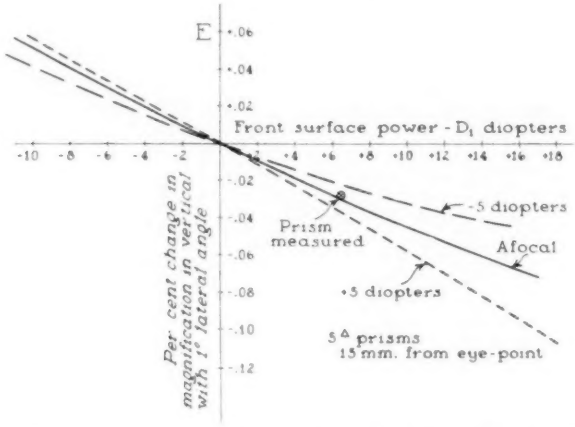


Fig. 7.—Magnitude of the change in vertical magnification with lateral angle (**E**) with different curvatures of the front surface, for afocal prisms and for +5 D. and —5 D. powers. These curves are for a 5△ prism placed 15 mm. from the eye point.

When an ophthalmic lens is decentered in order to obtain a prismatic correction, the same distortions as described previously exist. The decentration $z$ is related to this analysis approximately by $z=R_2 \sin \theta$, whence $k=z(R_1+R_2+t_o)/R_1R_2$. The apparent curvature of the images of lines at right angles to the direction of displacement of an ordinary spherical lens can be easily seen, especially as one looks through the lens near the edge.

It should be clear at this point that it is impossible to make an ophthalmic prism distortionless. We can eliminate the unsymmetric angular magnification in the base-apex meridian by a suitable choice of base curve, of the order of 9 D. as a round figure. We can eliminate the change in vertical angular magnification with

9. Hardy, L. H.: Clinical Use of Ophthalmic Prisms (Metric), Arch. Ophth. **34**:16-23 (July) 1945.

lateral angle by making the front surface of the prism a plane ($D_1$=0). The apparent curvature of the images of vertical lines seen through the prism cannot be eliminated under any circumstance.

## SUMMARY

In addition to the over-all angular magnification of an ophthalmic prism, the distortion is discussed under three aspects: (1) the unsymmetric angular magnification in the base-apex meridian; (2) the curvature of the images of vertical lines, those parallel to the base (perpendicular to the base-apex meridian), and (3) the change in the angular magnification in the vertical meridian with lateral angle. The last causes images of lines parallel to the base-apex meridian to appear to slant toward the apex. The angular magnification and distortion depend on the physical dimensions of the prism and on the distance of the prism from the eye. The distortion of the retinal image will therefore be different from the effect on movements of the eye behind the prism. The quantitative aspects of the distortion are indicated, and the conditions under which this distortion may be eliminated are discussed. The unsymmetric angular magnification can be eliminated by choice of suitable base curve; the change in angular magnification in the vertical meridian with lateral angle can be eliminated by making the front-surface curve a planocurve. The apparent curvature of the images of lines parallel to the base cannot be eliminated under any circumstance by change in base curves. It is concluded that distortionless ophthalmic prisms cannot be made by suitable flexure.

# CATARACT EXTRACTION AFTER GLAUCOMA SURGERY

## Laterointerior Approach Following Filtering Glaucoma Operation

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THE EXTRACTION of a cataractous lens from an eye involved by glaucoma of such severity that surgical control has been required presents many problems to the surgeon. In such cases the patient's future vision rests upon the surgical removal of a cataract from an eye whose metabolism has already been impaired by glaucoma and an antiglaucoma operation.

Considerations regarding the simultaneous management of a mature cataract and primary or secondary glaucoma which cannot be controlled medically are outside the province of this paper.

In such cases, three locations have been suggested for the corneal section: (1) in the upper limbus, directly through the bleb; (2) through the clear cornea anterior to the bleb, and (3) at the limbus laterally and inferiorly, the incision avoiding the bleb. Each of these locations has been proposed by various ophthalmologists, and the advantages and disadvantages of each method have been described.

Buffington [1] and Judd [2] each advocated a section through the filtration area. In Snip's [3] series some eyes were lost from uncontrolled glaucoma after this incision had been used. Kirby [4] recommended it only if the intraocular tension was 8 mm. (Schiøtz) or lower. McLean [5] sectioned the cornea anterior to the bleb and used his preplaced sutures in a groove to secure good apposition. Guyton [6] reported a series of nine cases in which the section had been made through the filtering bleb, with a subsequent operation necessary in only one. However, he recommended also that the section be made in the cornea below the bleb unless the intraocular tension was low.

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Read before the Section on Ophthalmology at the One-Hundredth Annual Session of the American Medical Association, Atlantic City, June 15, 1951.

1. Buffington, W. R.: Etiology and Treatment of Glaucoma Preceding and Following Cataract Extraction, Texas J. Med. **35**:700-704, 1939-1940.

2. Judd, J. H., in discussion on Guyton, J. S.: Choice of Operation for Primary Glaucoma Combined with Cataract, Tr. Am. Acad. Ophth. **49**:216-224, 1944.

3. Snip, R. T.: Cataract Extraction Following Filtering Operation, Wilmer Residents Association Program, 1949.

4. Kirby, D. B.: Prevention and Handling of Complications Arising During and After Cataract Extractions, Arch. Ophth. **25**:866-901 (May) 1941.

5. McLean, J.: Cataract Extraction After Glaucoma Operation, Am. J. Ophth. **25**:192-193, (Feb.) 1942.

6. Guyton, J. S.: Choice of Operation for Primary Glaucoma Combined with Cataract, Tr. Am. Acad. Ophth. **49**:216-224, 1944.

Van der Straeten [7] described the advantages of temporal section in the cornea anterior to the bleb. Gifford [8] applied van der Straeten's temporal approach in seven cases. He removed five cataractous lenses after iridencleisis and two after trephination. In three of the seven eyes the extraction had been intracapsular and in four extracapsular. Vitreous was not lost. In all seven eyes the intraocular tension remained controlled after removal of the cataract, but whether a miotic was required to assist in this procedure was not mentioned. Gifford reported that useful vision was regained in all eyes, and he attributed this to the fact that by chance none of them had been very severely damaged by the antecedent glaucoma.

Snip collected 42 cases of cataract extractions following filtering operations for glaucoma performed at the Wilmer Ophthalmological Institute between January, 1927, and July, 1948. They were grouped as follows: In 23 cases the section was made through the filtering area; in 11, in clear cornea anterior to the bleb; in 8, temporally or below, away from the filtering area. From an analysis of the results in these cases, he drew the following conclusions: 1. Cataract surgery should be performed anterior to or away from the filtering area, especially if the glaucoma operation has been an iridencleisis. 2. An intracapsular cataract extraction with no loss of vitreous improves the prognosis for continued function of a filtering bleb. 3. There is no significant difference in the incidence of corneal dystrophy in any method of cataract section studied. 4. An iridencleisis is less likely to require further operations for the control of glaucoma after cataract extraction than is a trephination.

The lateroinferior approach was used in each of 15 cases. We avoided making the section through the trephine bleb because of possible interference with its function, and because in several cases prior to this series unfortunate end-results had followed section through the bleb. We did not make the section in the clear cornea anterior to the bleb because of the difficulty of removing a lens in its capsule from under an overhanging shelf and the fear of corneal scarring in the pupillary area.

As detailed in the case reports (Table 1), the glaucoma was primary in each of the 15 cases and had been present for some time and operated upon before the lens became completely cataractous. The interval of time between the antiglaucoma surgery and the cataract extraction varied from 4 months to 11 years. The severity of the glaucoma is indicated by the fact that in 10 cases the fellow eye was blind from absolute glaucoma.

The technique of the operative procedure is illustrated in Figures 1 to 6.

## ANALYSIS OF RESULTS (Table 2)

*Improvement of Vision.*—The many variable factors in visual improvement defy generalizations and in each case must be considered individually. The most important factor which determines the final visual acuity is the degree to which the eye has been damaged by the glaucoma. The method of approach used, whether the intracapsular or the extracapsular, and similar considerations are important, but it

7. van der Straeten: Bull. Soc. belge opht. **52**:29, 1926.

8. Gifford, S. R.: Lens Extraction Following Filtering Operations for Glaucoma, Am. J. Ophth. **26**:468-473, 1943.

TABLE 1.—*Data on Cataract Extraction After*

| Initial | Age | State of Fellow Eye | Preoperative Condition of Eye | Type of Glaucoma Surgery and Date |
|---|---|---|---|---|
| A | 77 | O. S.: absolute glaucoma | Unknown | 1940: trephination-sclerectomy |
| A | 70 | O. D.: removal for complications following cataract extraction, 1947 | Tension O. S. 28-33 mm. despite miotics | 10/18/48: C. S.* trephination 11:00 limbus with peripheral iridectomy |
| B | 48 | O. D.: Normal visual acuity 20/20 | Tension O. S. 35 mm. despite miotics | 11/29/46: C. S. trephination with peripheral iridectomy |
| B | 79 | O. S.: enucleation for absolute glaucoma, 1946 | Unknown | Aug. 1945: C. S. trephination |
| C | 60 | O. D.: enucleation for absolute glaucoma, 1946 | Unknown | 1943: C. S. trephination |
| C | 62 | O. S.: enucleation for absolute glaucoma | Tension O. D. 35 mm. despite miotics | 8/14/46: C. S. trephination |
| F | 79 | O. D.: enucleation for absolute glaucoma, 1946 | Unknown | 1937: C. S. trephination |
| J | 74 | O. S.: glaucoma; visual acuity 20/100 | Tension 35 mm. despite miotics | 7/26/48: C. S. trephination with peripheral iridotomy |
| J | 82 | O. S.: absolute glaucoma | Tension 32 mm. despite miotics | 9/17/47: C. S. trephination |
| M | 64 | O. S.: chronic noncongestive glaucoma, controlled with miotics | Tension 35 mm. despite miotics | 8/6/48: C. S. trephination |
| P | 64 | O. S.: enucleation for absolute glaucoma | Tension 35 mm. despite miotics | 8/19/48: C. S. trephination |
| R | 67 | O. D.: glaucoma controlled with c. s.* trephination 4/8/43 | Tension 35 mm. despite miotics | 2/5/44: C. S. trephination |
| S | 78 | O. D.: absolute glaucoma; eye enucleated | Tension 30 mm. despite miotics | 6/9/48: C. S. trephination |
| W | 70 | O. S.: eye enucleated, 1948, for absolute glaucoma | Tension 52 mm. despite miotics | 4/18/49: C. S. trephination |
| W | 63 | O. S.: eye enucleated for absolute glaucoma | Tension 35 mm. despite miotics | 9/12/47: C. S. trephination with an iridotomy<br>Note: All C. S. trephination at 12:00 on limbus unless otherwise noted |

* C. S. indicates "corneoscleral."

must not be forgotten that the surgeon begins with whatever has been left by the glaucomatous process.

*Control of Intraocular Tension.*—If the cataractous lens is removed in the capsule without loss of vitreous, successful control of the intraocular tension without resort to miotics or surgery is more likely. However, occasionally either or both may be needed, even when these surgical objectives have been achieved.

In the three cases in which vitreous presented or a small amount was lost, diisopropyl fluorophosphate was required to keep the intraocular tension under control. In none of the 15 cases was it impossible to control the intraocular tension after the extraction of the cataractous lens.

*Surgical Treatment of Glaucoma After Cataract Extraction.*—In 2 of the 15 cases a surgical procedure was required to assist in controlling the intraocular tension after the trephination. In one, a cyclodialysis controlled the intraocular tension, and vision remained at 20/40. In the other, a cyclodialysis, an iridencleisis, a

*Filtering Glaucoma Operation in Fifteen Cases*

| Postsurgical Course | Date of Cataract Surgery | Details of Cataract Surgery | Surgery After Cataract Extraction<br>Miotics Required | Findings at Last Follow-Up Observation |
|---|---|---|---|---|
| Glaucoma controlled without miotics | 2/21/49 | Intracapsular, after posterior synechiae severed; with Kirby forceps; large bead of vitreous lost | Retinal reattachment not attempted because of gallbladder disease<br>None | All vision lost |
| Glaucoma controlled without miotics | 2/21/49 | Extracapsular; Arruga forceps; vitreous presented; none lost | .....<br>Diisopropyl fluorophosphate | 4/6/41: vision 20/60; tension 24 mm. Hg |
| Glaucoma controlled without miotics | 6/28/48 | Intracapsular; no loss of vitreous | Cyclodialysis after cataract extraction<br>Pilocarpine and physostigmine | 6/16/49: vision 20/40; tension 23 mm. Hg |
| Glaucoma controlled without miotics | 11/29/48 | Extracapsular; Arruga forceps; no loss of vitreous | ..... | 5/10/51: vision 20/25; tension 23 mm. Hg |
| Glaucoma controlled with diisopropyl fluorophosphate | 4/15/49 | Intracapsular; Kirby forceps; no loss of vitreous | Carbachol benzalkonium<br>.....<br>Diisopropyl fluorophosphate | 4/25/51: vision, counting fingers 3 ft.; tension 25 mm. Hg |
| Glaucoma controlled without miotics | 1/ 3/47 | Extracapsular; Arruga forceps; small loss of vitreous | Cyclodialysis, iridencleisis, and trephination after cataract extraction<br>Diisopropyl fluorophosphate | 11/3/49: vision 20/20; tension 26.5 mm. Hg |
| Miotics required intermittently | 1/25/50 | Intracapsular; Bell erisophake; no loss of vitreous | Sphincterectomy for vision planned, but patient died<br>..... | 5/3/50: vision, hand movements; tension 20 mm. Hg |
| Miotics required intermittently | 3/14/49 | Extracapsular; Arruga forceps; no loss of vitreous | .....<br>Diisopropyl fluorophosphate | 4/3/51: vision 20/100; tension 23 mm. Hg |
| Glaucoma controlled without miotics; dense posterior synechiae developed | 8/30/48 | Intracapsular; Arruga forceps; no loss of vitreous | Sphincterectomy for vision<br>..... | 4/11/51: vision, hand movements 2 ft.; tension 25 mm. Hg |
| Glaucoma controlled with diisopropyl fluorophosphate | 1/19/51 | Intracapsular; Bell erisophake; no loss of vitreous | .....<br>Diisopropyl fluorophosphate | 5/15/51: vision 20/20; tension 22 mm. Hg |
| Glaucoma controlled without miotics | 9/27/48 | Intracapsular; Arruga forceps; no loss of vitreous | .....<br>..... | 12/19/50: vision 20/25-2; tension 22 mm. Hg |
| Glaucoma controlled without miotics | 5/18/48 | Intracapsular; Arruga forceps; no loss of vitreous | Sphincterectomy for vision<br>..... | 4/11/50: vision 20/30; tension 15 mm. Hg |
| Glaucoma controlled with pilocarpine; intumescence of lens noted 10/7/48 | 10/23/48 | Intracapsular; Arruga forceps; no loss of vitreous | .....<br>Diisopropyl fluorophosphate | 2/8/51: vision 20/60; tension 20 mm. Hg |
| Glaucoma controlled without miotics | 12/ 2/49 | Intracapsular; Bell erisophake; no loss of vitreous | Sphincterectomy for vision<br>Diisopropyl fluorophosphate | 1/10/51: vision 20/70-2; tension 25 mm. Hg |
| Glaucoma controlled with pilocarpine | 7/11/49 | Extracapsular; expressor and loop; no loss of vitreous | .....<br>..... | 8/31/50: vision 20/60; tension 20 mm. Hg |

TABLE 2.—*Effect of Loss and Nonloss of Vitreous (Series of 15 Eyes)*

| | No. of Eyes | Effect on Vision | Postsurgical Course |
|---|---|---|---|
| No Loss | 11 | (2) 20/20; (2) 20/25; (1) 20/40, (2) 20/60; (1) 20/100; (1) f. c. 8'; (2) h. m. | Controlled without miotics, 5<br>Controlled with miotics, 5<br>Controlled with miotics and cyclodialysis, 1 |
| Vitreous presented or small amount lost | 3 | 20/25; 20/60; 20/70-2 | Controlled with miotics, 2<br>Controlled with miotics, trephination and cyclodialysis, 1 |
| Large bead of vitreous lost | 1 | Late retinal detachment | Hypotony |

trephination, and continuous use of diisopropyl fluorophosphate were required. Corrected vision remained 20/25. In neither case did the loss of visual field progress.

However, in Snip's series of 42 cases, in which three different antiglaucoma procedures were employed, nine eyes required further operation for glaucoma after

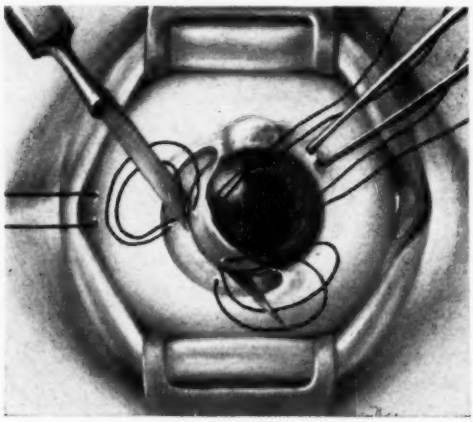

Fig. 1.—After a bridle suture has been placed through the lateral rectus insertion to assist in fixing the position of the eye, a limbus-based conjunctival flap about 3 mm. wide is constructed. It is extended from about 11 to 5 o'clock at the limbus on the right eye, or from about 1 to 7 o'clock on the left eye. Since the horizontal diameter of the cornea is 1 mm. greater than the vertical, a lateral corneal section must extend slightly further around the limbus to permit the removal of the lens. Beneath the conjunctival flap, at the limbus, a groove about one-half the thickness of the sclera is made with a sclerotome, the position of the eye being steadied with a pick. This permits the use of the McLean appositional sutures and serves as a guide for the completion of the corneal section.

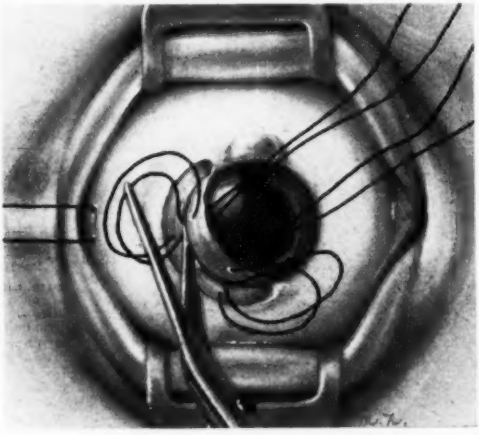

Fig. 2.—Firm fixation of the eye is obtained by grasping the conjunctiva and sclera with scleral fixation forceps near the limbus opposite the midpoint of the intended corneal section. The von Graefe knife is inserted into the anterior chamber through the groove near one suture and is directed across the anterior chamber and out in the groove at a point near the other suture. During this section, the knife blade is held at such an angle that the corneal shelf is made as small as possible.

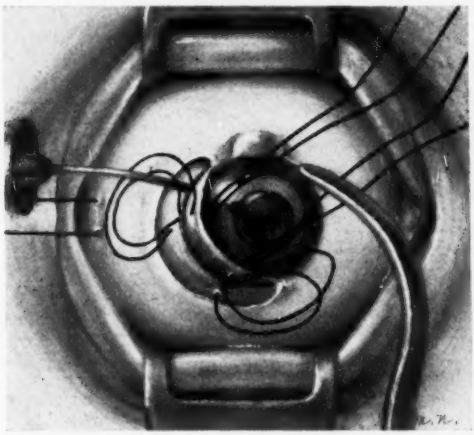

Fig. 3.—The corneal section is enlarged with corneal scissors. If direct inspection of the lens indicates intumescence or hypermaturity, the corneal section may be extended further beneath the conjunctival flap to facilitate its removal.

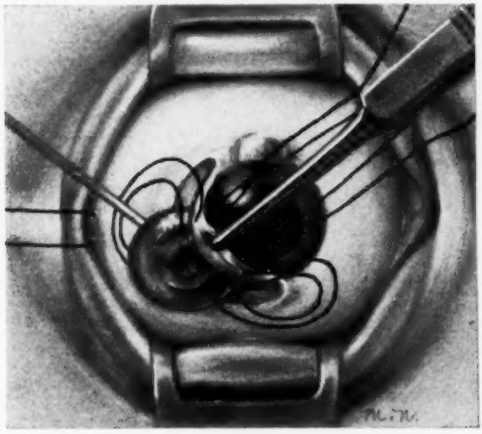

Fig. 4.—The Bell erisophake is filled with isotonic sodium chloride solution and inserted into the anterior chamber. As it is being maneuvered into position against the anterior capsule, the rubber bulb is slowly and firmly squeezed to irrigate the anterior chamber of any blood clot or pigment and to push the iris border away from the erisophake tip. When this is placed in contact with the anterior capsule, the pressure on the bulb is slowly released, so that the lens capsule is grasped midway between the center of the lens and the equator in the upper nasal quadrant. Point pressure is then exerted with the Budapest expressor at the upper nasal quadrant of the limbus, the lens being held firmly and gently tilted upward at this point with the Bell erisophake. The zonular membrane is ruptured in the upper nasal quadrant.

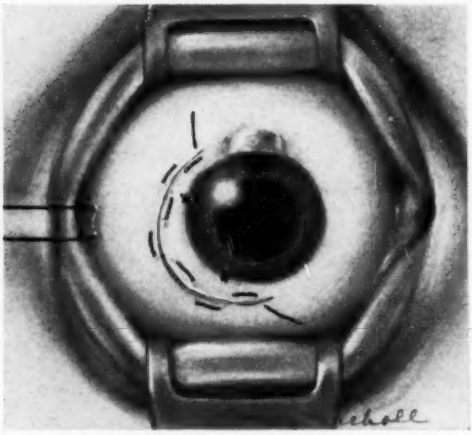

Fig. 5.—As the upper nasal edge of the lens becomes more elevated, the zonular membrane is separated further along each side. As the nasal edge of the lens is tumbled so that the lens will present in the corneal section, the Budapest expressor is used to push the lens along through the corneal flap.

At this point, ask the experienced assistant to hold the conjunctival flap lightly near the cornea at the midpoint of the incision. It has been stated many times that if there is no external pressure against the eye the vitreous will not flow out; but, unfortunately, it is not always possible to remove all external forces, even with a canthotomy and the best of specula.

If the vitreous does threaten to prolapse, the assistant holds the corneal edge against the lens as it is being delivered and immediately thereafter holds the cornea firmly against the sclera until the surgeon can draw the loops out of the sutures and tie them.

If the iris is in contact with the posterior surface of the cornea after the corneoscleral sutures have been tied, a small amount of isotonic sodium chloride solution is inserted with an irrigator to separate them and to maintain the chamber angle for filtration.

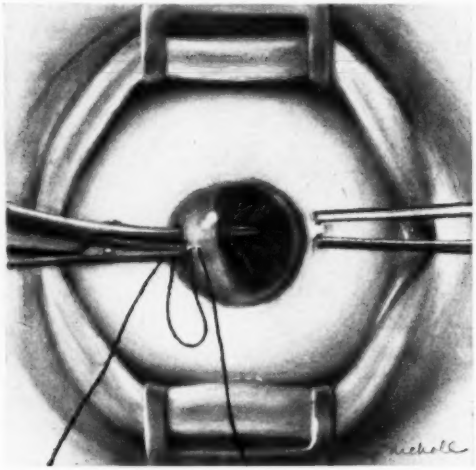

Fig. 6.—A continuous mattress suture is laced into the conjunctival edges to hold them together, after the corneoscleral sutures have been tied. The conjunctival suture is allowed to remain for one week and is then removed. The corneoscleral sutures are removed after two weeks. The correcting lens is usually fitted six weeks after the operation has been performed.

the cataract extraction. Eight of these eyes had trephinations and one had an iridencleisis.

*Operation for Improvement of Vision After Cataract Extraction* (Four Eyes).—In two eyes in which the lens was delivered intracapsulary, the iris prolapsed against the cornea (not through the wound) and pulled over to the limbal wound so far that the pupillary opening was closed. Corneal opacity, endothelial and stromal, developed at the site of contact. An incision was made in the opaque area of the cornea, near the limbus, with a small keratome. The iridocapsulotomy scissors were introduced through this opening and inserted into the iris. The iris was sectioned so that a clear area of the cornea could be utilized for vision (Fig. 7).

In two other cases, without anterior synechiae, a corneal opacity developed in the limbal area along the line of incision, and this extended into the pupillary area; a sphincterotomy was indicated at the appropriate point to increase the pupillary aperture and to permit vision through a clear area of the cornea.

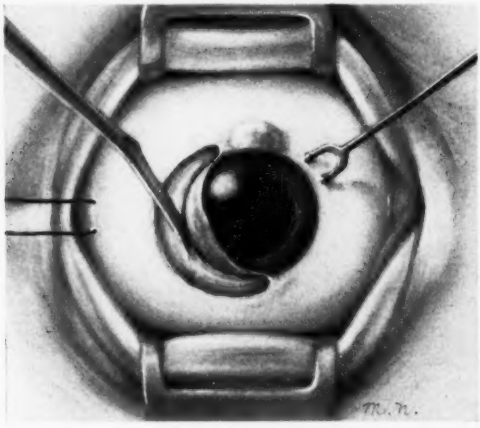

Fig. 7.—Iridotomy for the complicated case in which the pupillary opening is closed off. An incision is made in the opaque area of the cornea, near the limbus, with a small keratome. The iridocapsulotomy scissors are introduced through this opening and inserted into the iris. The iris is sectioned so that a clear area of the cornea can be utilized for vision.

## COMMENT

We prefer the lateroinferior corneal section because with this method there is less probability of interference with the antiglaucoma operation. In only 2 of the 15 eyes was a surgical procedure required to control glaucoma after the cataract extraction. However, the greatest disadvantage of this method is that in some instances it is followed by derangement of corneal metabolism, perhaps because the ability of the cornea to free itself of fluid is reduced. In four eyes corneal opacification (a more proper term for the condition than corneal dystrophy) extended toward the center of the cornea sufficiently to require an iridotomy on the opposite side to allow vision through a clear area. Sternberg and Meyer [9] have recently described

9. Sternberg, P., and Meyer, S. J.: Corneal Dystrophy Following Secondary Intraocular Operations, A. M. A. Arch. Ophth. **46**:527-530 (Nov.) 1951.

cases in which corneal dystrophy followed secondary intraocular operations performed in a different quadrant. They suggest that the cause is the additional damage to the endothelium.

Every attempt should be made to remove the cataractous lens in the capsule without loss of vitreous; yet even when this objective is attained, miotics or another antiglaucoma operation may be required. Failure to achieve this objective will not result in loss of the vision, but usually miotics are required.

SUMMARY

The surgical management of a series of 15 cases of cataract extraction, which was required in eyes that had been operated on for glaucoma, is presented, the technical details being included. A final conclusion that this is the best method possible cannot be drawn from these cases, because of their relatively small number and because of the difficulties that attend the method. The findings are presented because of the few self-critical articles available on the subject and because one may review this material for comparison with one's own technique or method of management of this difficult problem.

# DEVELOPMENTAL ANATOMY OF NASOLACRIMAL DUCT

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AS A PART of the scientific exhibit of the American Academy of Ophthalmology and Otolaryngology, in 1950, I reviewed the developmental anatomy of the nasolacrimal duct. At that time, and since, I have been repeatedly asked for reprints of, or references to, the literature on this subject. Although much of the material used in the exhibit has been published in the anatomic, embryologic, and ophthalmologic literature, some of it is original, and the remainder bears repetition.

The nasolacrimal passageway arises from a thickening of the ectoderm in the naso-optic fissure. The thickened ectoderm buries itself in the mesenchyme between the lateral nasal process and the maxillary process, detaches itself from the surface, and extends downward. Later, the epithelial cord canalizes and becomes patent at the ocular end and, finally, at its lower nasal end. The latter, the nasal connection, may often not be completed at birth.

In the 7-mm., or 32-day, embryo a thickening of the surface ectoderm appears in the rudimentary naso-optic fissure (Fig. 1). This thickened ectoderm lies between the lateral nasal process and the maxillary process, at about the same time that the olfactory groove is found (Fig. 2*A*). By the 9-mm., or 37-day embryonic stage, the olfactory pit is well formed, and the surface ectoderm of the naso-optic fissure is thick enough to be well differentiated (Fig. 2*B*). As the ectoderm thickens, it begins to project down beneath the surface (Figs. 3 and 4*A*). This can be seen in the 12-mm., or 42-day, embryo. Caudal and cephalic branchings of the epithelial cord occur early (Fig. 4*B*). These form the upper and lower extensions of the lacrimal passageways, the upper extension forming the cupula and canaliculi, the caudal projection forming the duct. In the 15-mm., or 44-day, embryo, the olfactory sac is well formed, and the ectoderm is definitely projecting and extending inward (Fig. 4*C*).

As the ectoderm extends downward, it detaches itself from the surface and buries itself in the mesenchyme (Figs. 5 and 6*A* and *B*). This epithelial cord (Figs. 7 and 8*A*) extends downward toward the nasal cavity and is entirely buried between the inferior meatus of the nasal fossa and the inner canthus of the eye (Fig. 8*B*). It later canalizes to become the lacrimal passageway. Beginning canalization is already occurring in the 32- to 36-mm., 60-day, or late, embryonic stage (Figs. 9 and 10*A* and *B*).

The prenatal development of the human body is divided into three main periods. The embryonic period begins after the third week and extends through the second month of prenatal life. The fetal period begins after the second month of gestation and extends until birth. In the embryonic period there is differentiation of the main systems and organs of the body; during the fetal period a rapid absolute increment

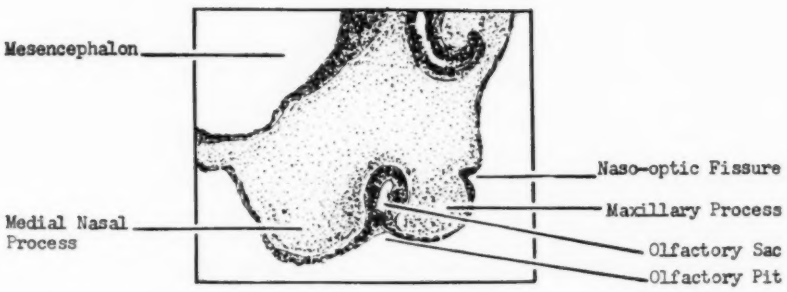


Fig. 1.—In the 7-mm. embryo a thickening of the surface ectoderm is first observed in the rudimentary naso-optic fissure. This later buries beneath the surface to become the epithelial cord of the nasolacrimal passageways.

| | |
|---|---|
| 7 mm. (32 day) | Kampmier Series 27, Slide 2 |
| 9.5 mm. (37 day) | Kampmier Series 23, Slide 4 |

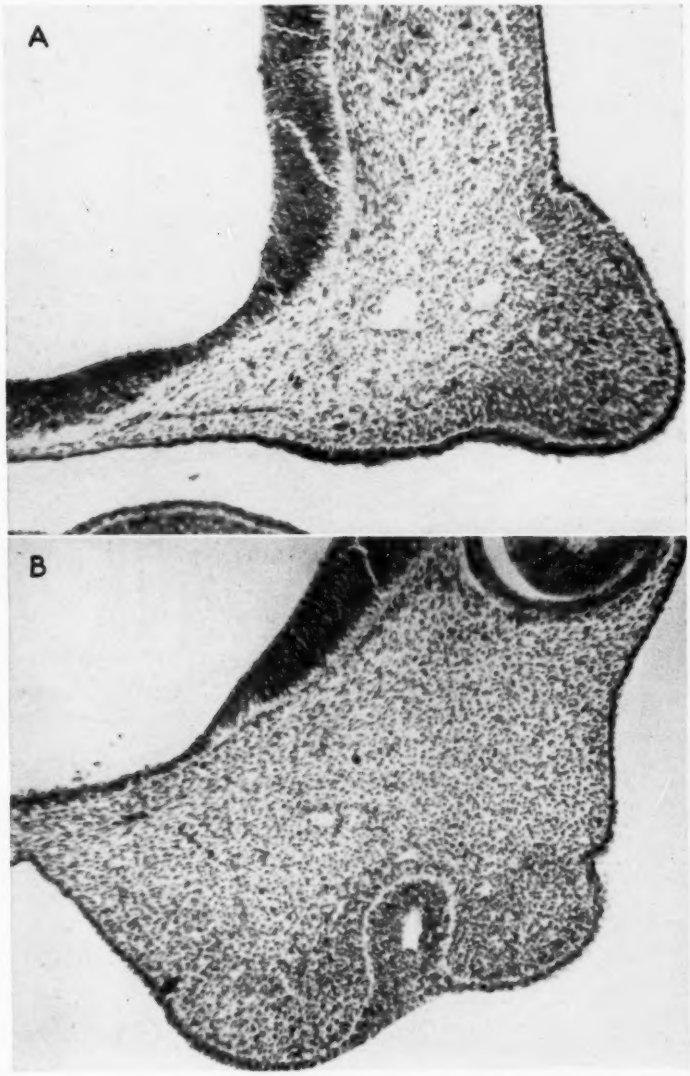


Fig. 2.—*A*, frontal section, 7-mm. (32-day) human embryo. Kampmier Series 27; Slide 2.

The prosencephalon surrounds the large space to the left. The indentation of the olfactory groove is seen below, between the medial nasal process and the maxillary process. The naso-optic fissure lies laterally, between the maxillary and the lateral nasal process.

*B*, frontal section, 9.5-mm. (37-day) human embryo. Kampmier Series 23; Slide 4.

The olfactory pit and the beginning olfactory sac lie between the medial nasal process and the maxillary process. The thickened ectoderm of the naso-optic fissure lies between the maxillary and the lateral nasal process. The optic cup is differentiating into its component parts.

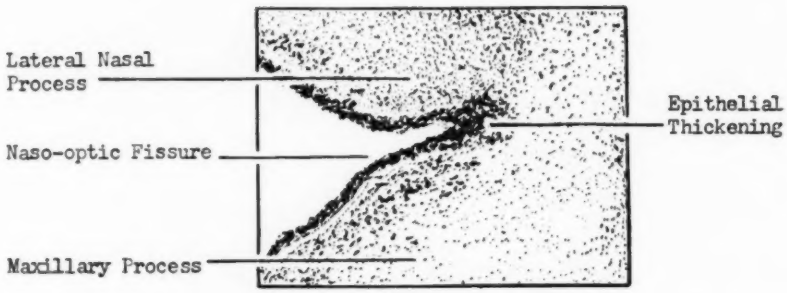


Fig. 3.—Region of rudimentary naso-optic fissure.

A thickening of the epithelium of the rudimentary naso-optic fissure buries itself in the mesenchyme. This cord grows cephalad to form the cupula of the lacrimal sac and the two lacrimal ducts.

| | |
|---|---|
| 12 mm. (42-43 day) | Arey Series |
| 15 mm. (44 day) | Kampmier Series; Slide 7 |
| 17 mm. (46 day) | Kampmier Series; Slide 8 |

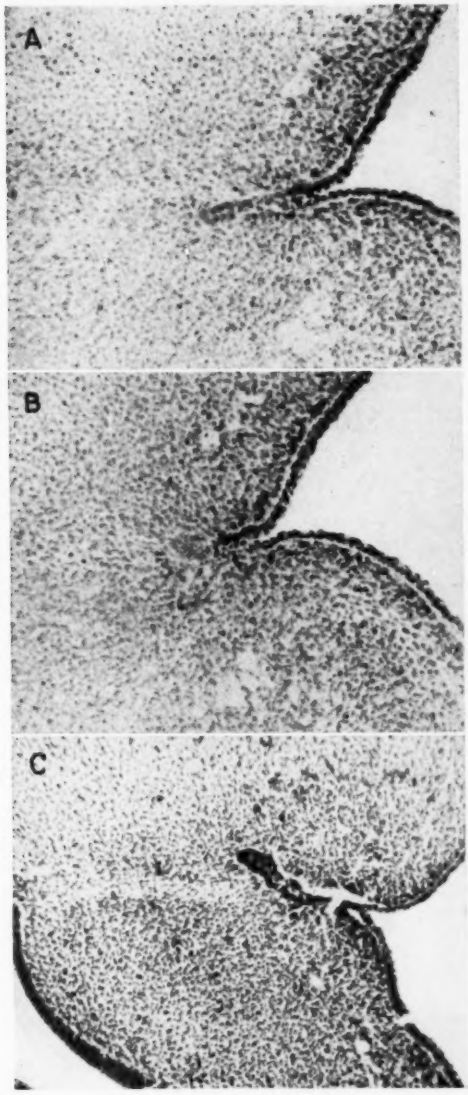


Fig. 4.—*A*, frontal section, 12-mm. human embryo. Arey Series.

The thickening of the surface ectoderm of the naso-optic fissure extends down into the mesenchyme between the maxillary process, below, and the lateral nasal process, above.

*B*, frontal section, 12-mm. (42-to-43-day) human embryo. Arey Series.

The cephalic and caudal branches of the buried ectoderm are seen in the mesenchyme between the maxillary and the lateral nasal process.

*C*, frontal section, 15-mm. (44-day) human embryo. Kampmier Series 26; Slide 8.

The nasal cavity is on the left, definite and well formed. The epithelial cord extends inward from the naso-optic fissure into the mesenchyme, separating the maxillary and the lateral nasal process.

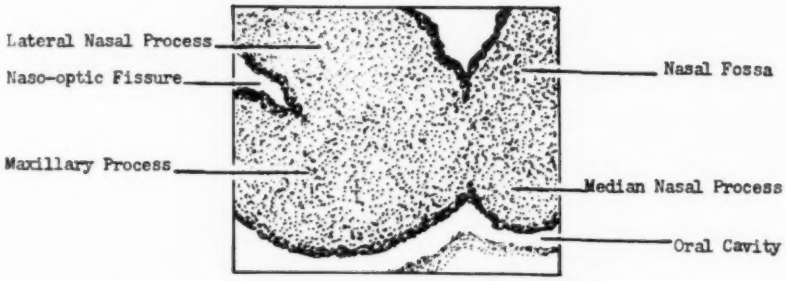


Fig. 5.—Detachment of the epithelium from its surface connections.

The strand of thickened epithelium proliferates and detaches from the surface connection. Surrounded by the subjacent mesenchymal tissue, it shrinks away from the surface epithelium and becomes the anlage of the nasolacrimal passageways.

| | |
|---|---|
| 13 mm. (43 day) | Kampmier Series; Slide 9 |
| 13 mm. (43 day) | Kampmier Series; Slide 8 |
| 14 mm. (43-44 day) | Kampmier Series; Slide 5 |
| 15 mm. (44 day) | Kampmier Series 26; Slide 8 |

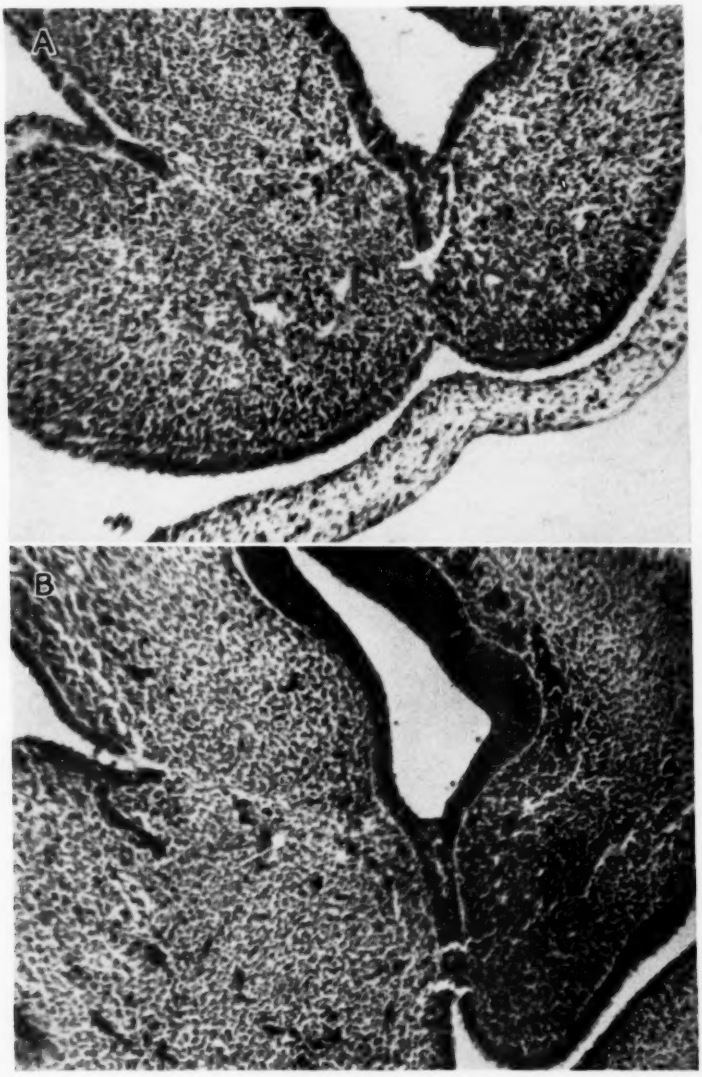


Fig. 6.—*A*, frontal section, 13-mm. human embryo. Kampmier Series 17, Slide 9.

The epithelial cord extends into the mesenchyme between the embryonic maxillary and the lateral nasal process. The olfactory groove is closing; the olfactory sac is well defined.

*B*, frontal section, 13-mm. (43-day) human embryo. Kampmier Series 18, Slide 8.

The cephalic and caudal divisions of the epithelial cord are shown extending toward the olfactory sac. The olfactory groove is not entirely closed.

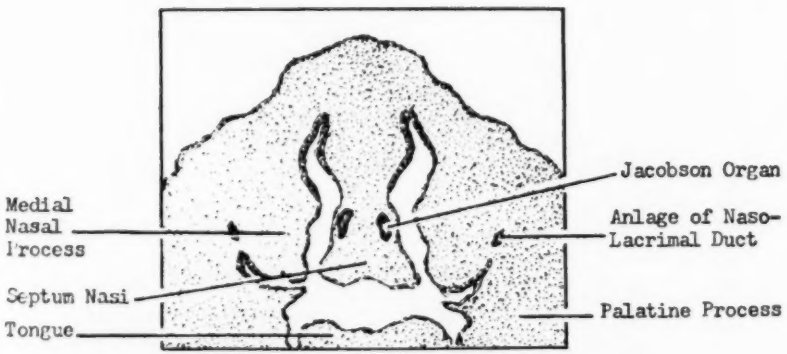


Fig. 7.—Nasalward extension of the nasolacrimal-duct anlage.

The buried epithelium grows nasalward from its surface attachment to connect with the inferior meatus and becomes the nasolacrimal duct.

| | |
|---|---|
| 20 mm. (48 day) | Arey Series |
| 20 mm. (48 day) | Arey Series |
| 26 mm. (50 day) | Kampmier Series 10; Slide 29 |
| 21 mm. (49 day) | Kampmier Series 9 |
| 25 mm. (50 day) | Cassady Series |

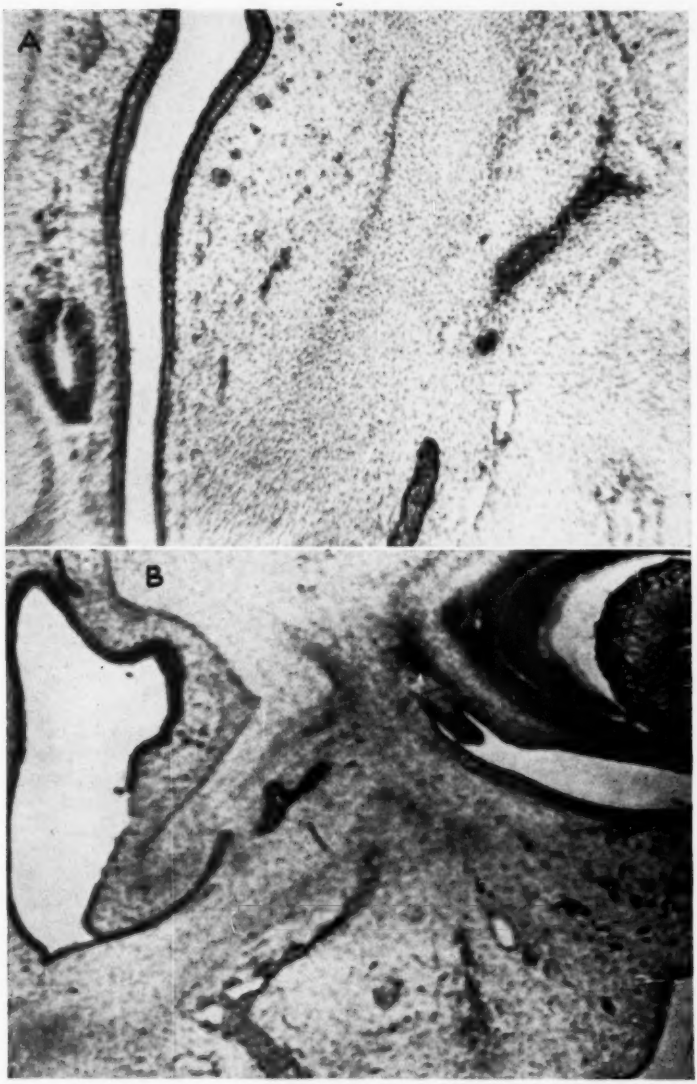


Fig. 8.—*A*, frontal section, 26-mm. (50-day) human embryo. Kampmier Series 10, Slide 29. To the left is the nasal septum, containing Jacobson's organ. The inferior turbinate and the epithelial fold of the inferior meatus are to the right. The buried epithelium of the nasolacrimal anlage nearly joins the epithelium of the nasal cavity.

*B*, frontal section, 25-mm. (50-day) human embryo. Cassady Series.

The nasal fossa, the epithelial fold under the inferior meatus, and the inner canthus of the eye, with the epithelial cord of the nasolacrimal duct between, are seen.

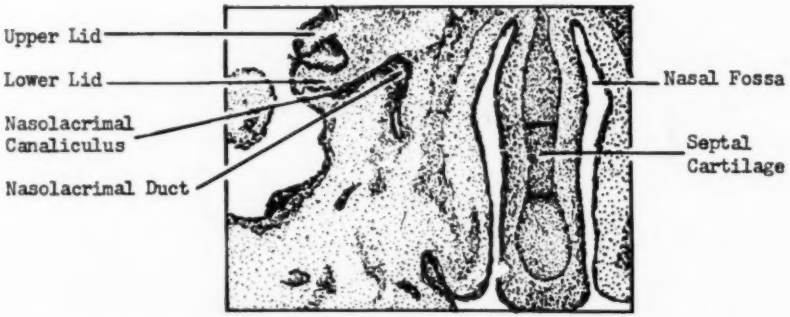


Fig. 9.—Segments of epithelial cord between the eye and the nose.

The upper and lower lids are shown with segments of epithelium extending downward to the inferior meatus of the nose. This later will canalize to form the nasolacrimal duct.

32 mm. Kampmier Series 15; Slide 44
32 mm. Kampmier Series 14; Slide 43
36 mm. Kampmier Series; Slide 44

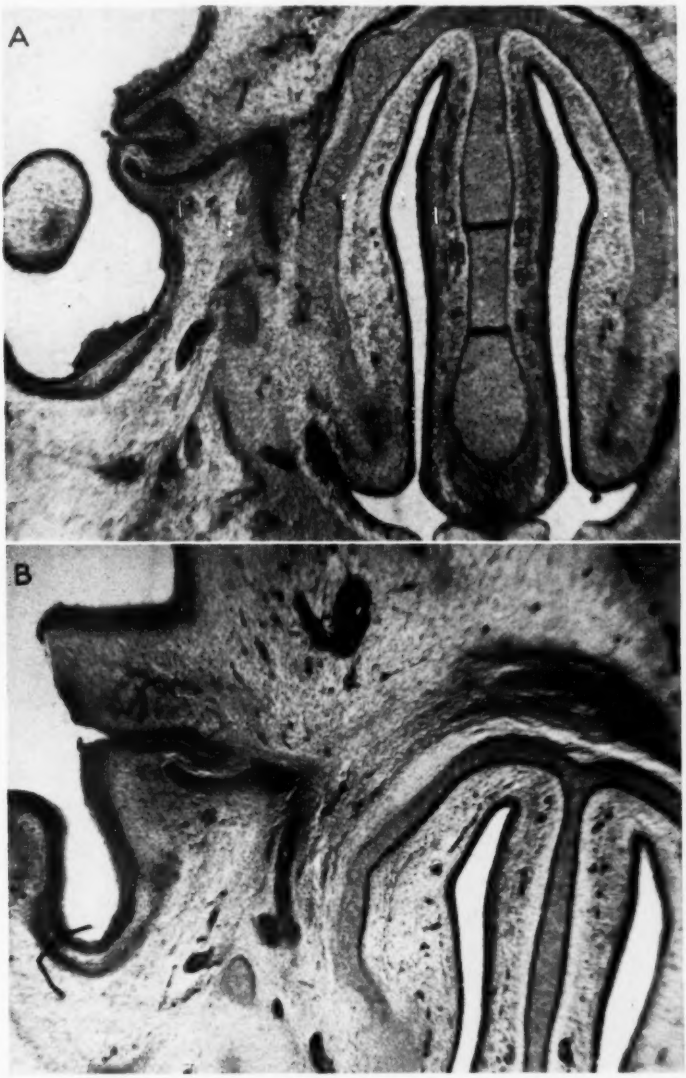


Fig. 10.—*A*, frontal section, 32-mm. (60-day) human embryo. Kampmier Series 15; Slide 44.

The eyelids, the nasal septum, and the nasal cavity are shown. The horizontal portion of the nasolacrimal anlage in the lower lid, as well as part of its vertical portion as it extends nasalward, is seen.

*B*, frontal section, 36-mm. (60-day) human embryo. Kampmier Series 14; Slide 44.

The horizontal and vertical portions of the lacrimal duct extend from the lower lid toward the nose.

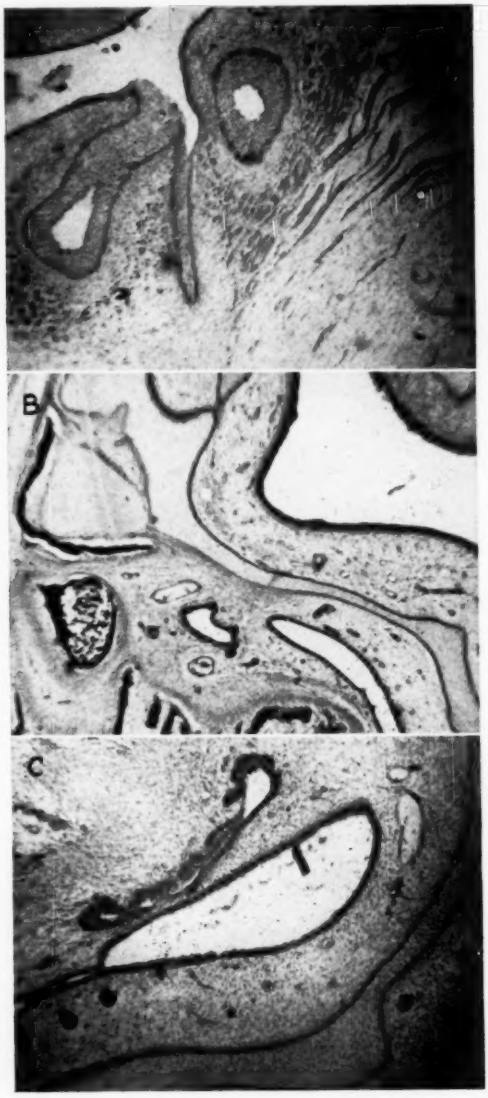


Fig. 11.—*A*, frontal section, 90-mm. (3-mo.) human fetus. Cassady Series.

The superior punctum and the superior and inferior canaliculi are well canalized.

*B*, frontal section, 95-mm. (3½-mo.) human fetus. Kampmier Series 24; Slide 13.

The inferior turbinate curves downward, separating the middle and the inferior meatus. A cross section of the lower end of the lacrimal duct lies close to the mucosa of the inferior meatus.

*C*, frontal section, 90-mm. (3-mo.) human fetus. Cassady Series.

The inferior meatus, lined with mucosa, is separated by a fairly thick membrane from the lower end of the nasolacrimal duct. The latter is partially canalized.

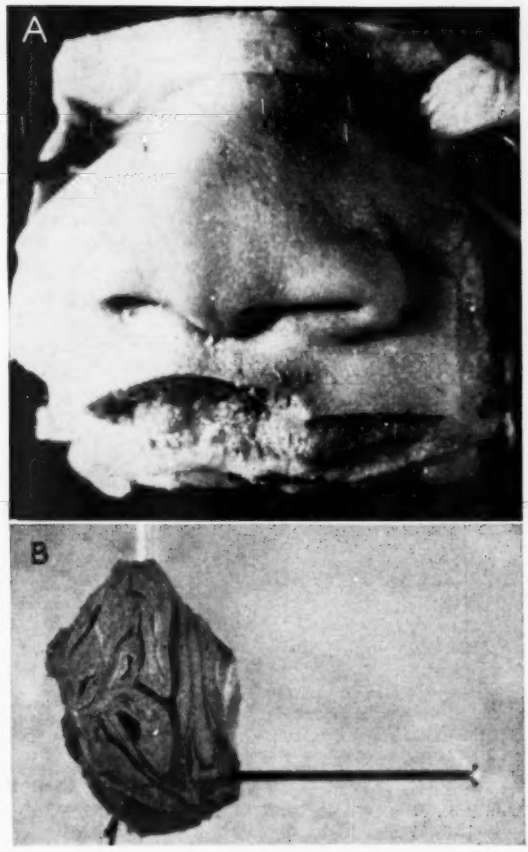


Fig. 12.—*A*, photograph of the block of the nose and lacrimal passages removed from one of the stillborn fetuses. Department of Embryology, Carnegie Institution of Washington, Fetus 5498-13.

*B*, photograph of one of the trimmed-down blocks of the nose and lacrimal passages, as it was ready for embedding before sectioning. The horizontal and vertical pins were embedded in the block for orientation in the correct plane for sectioning. This specimen was from the Department of Embryology, Carnegie Institution of Washington, Fetus 7525-23R.

and a marked variance in the relative growth rates of different segments and parts of the body are apparent. During the early increment period the upper and lower lids are differentiated, and segments of epithelium extend downward toward the inferior meatus of the nose.

In the early fetal stage the segmental canalization of the duct becomes definite. It begins at the upper (ocular) end (Fig. 11*A*) and progresses downward (Fig. 11*B*). The horizontal portions of the duct establish lumina before the vertical portions. The canalization is a rearrangement of the epithelial cells around a centrally placed lumen (Fig. 11*C*). During the fetal period there is gradual extension of the canalization until the entire duct is patent. The ocular lumen connects with the inferior and superior canaliculi and with the surface of the lid borders. At birth the lower end of the lacrimal duct is not usually open to connect with the nasal cavity. The point of coalescence between the nasal end of the nasolacrimal duct and the mucous membrane of the inferior meatus is the last point in the duct system to become patent. In most instances, canalization here is deferred until postnatal

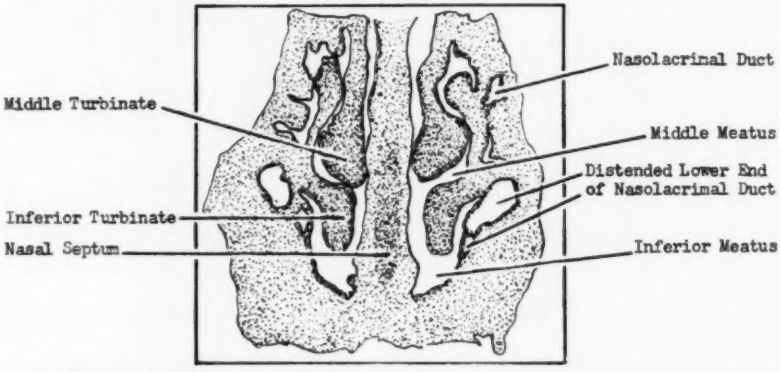


Fig. 13.—Nasal end of the nasolacrimal duct.

The nasal end of the nasolacrimal duct in the majority of instances is not patent at birth. Serial sections through this region and the injection of the nasolacrimal duct through the upper and lower canaliculi showed the duct was not patent into the nose. Serial sections showed a distended lower end of the duct.

Full-term stillborn male infant 5716, Sections 150, 160, 170, and 180, Department of Embryology, Carnegie Institution of Washington.

life. In a majority of very young infants there is a membranous barrier between the nasolacrimal duct and the inferior meatus, so that the lumen of the duct does not open into the nose at birth.

## PRESENT STUDY

The high incidence of dacryocystitis in infancy would attest to there being an interference with the lacrimal drainage in early infancy. In 1950, interest in whether the lacrimal duct was patent occasioned the examination of a number of full-term stillborn fetuses to determine how often the lacrimal duct is impatent at birth. The most complete collection of fetuses is that of the Department of Embryology, the Carnegie Institution of Washington. With the assistance and cooperation of Dr. George W. Corner, Director, the nasolacrimal passages of 15 full-term stillborn infants were examined at the Institution.

The fetuses were softened as much as possible, and then the upper and lower canaliculi were injected, first, with water and then with India-ink solution. The patency of the ducts

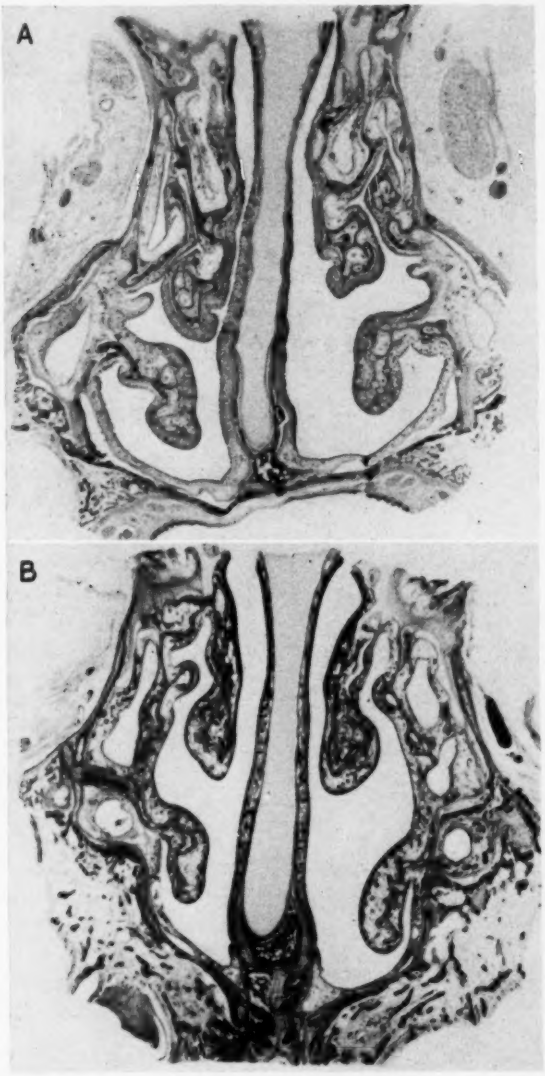


Fig. 14.—*A*, frontal section, lower end of the nasolacrimal duct of full-term stillborn infant 6415, Carnegie Institution of Washington.

The nasal cavity is separated from the lower end of the lacrimal duct by a thick membrane.

*B*, frontal section, lower end of the lacrimal duct of full-term stillborn infant 6724, Department of Embryology, Carnegie Institution of Washington.

The lower end of the lacrimal duct is separated from the inferior meatus of the nose by a thick membrane.

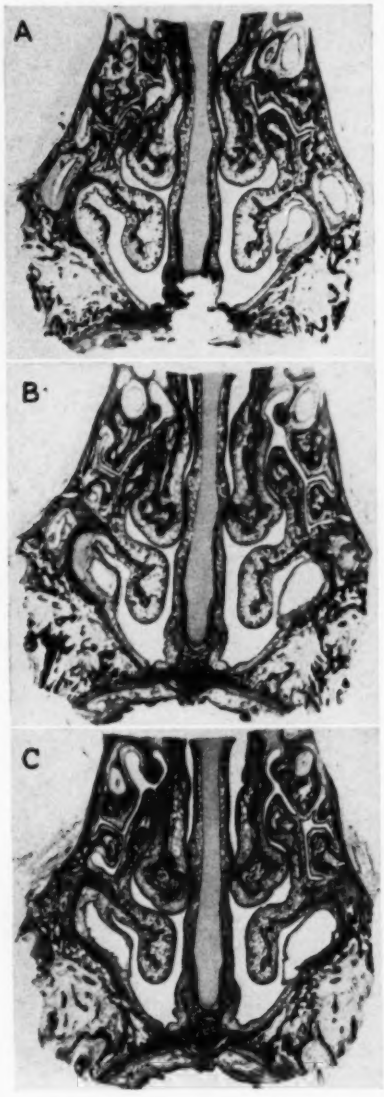


Fig. 15.—*A, B, C,* frontal sections, lower end of the lacrimal duct of full-term stillborn infant 5716; Sections 150, 160, and 180.

As the serial sections, taken 10 sections apart, are traced backward, the lower end of the lacrimal duct becomes thinner, but is still separated from the inferior meatus of the nose by a membrane.

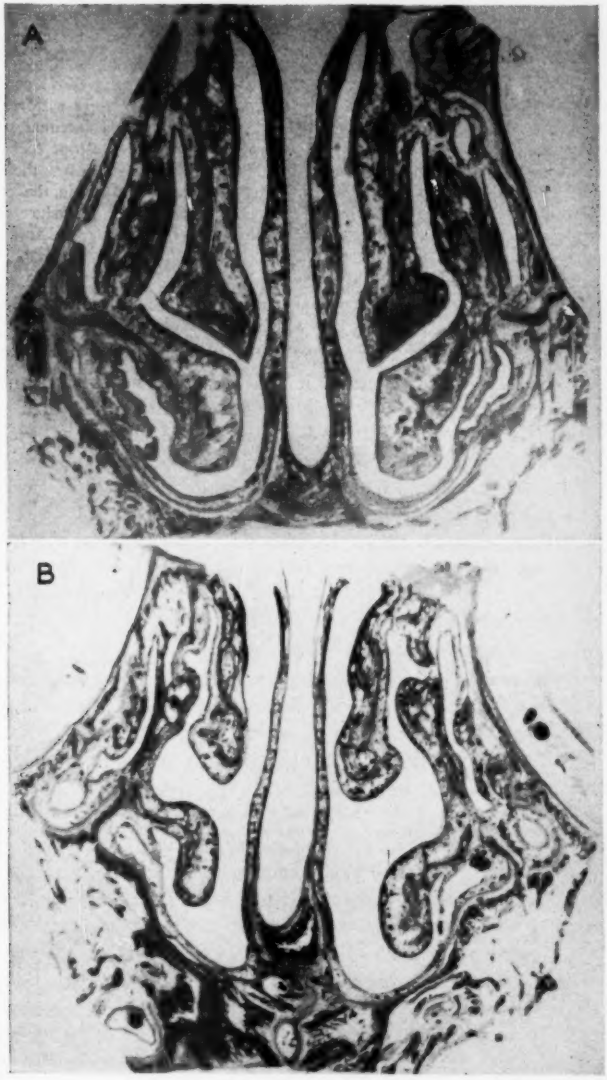


Fig. 16.—*A*, frontal section, lower end of the lacrimal duct of stillborn twin female infant 5498-13; Section 281, Department of Embryology, Carnegie Institution of Washington.

The lower end of the nasolacrimal duct is not patent but is still separated from the nasal fossa by the thickness of two abutting mucous membranes, that of the duct and that of the inferior meatus of the nasal fossa.

*B*, frontal section, lower end of the lacrimal duct of stillborn boy, aged 38 wk., Infant 7195, Department of Embryology, Carnegie Institution of Washington.

In this section the membrane which separates the lower end of the duct from the nasal cavity is very thin, but its patency had been tested with an India-ink solution by pressure injected into the upper and lower canaliculi.

was tested by use of alternate pressure and release for two or three minutes to force the solution through the ducts into the nose. If there was no opening into the nose, the sac and lower end of the duct became distended and filled with the India-ink solution.

After the patency of the duct was tested by this method, a block (Fig. 12*A*) containing the nose, the inner portion of the eyelids, and both lacrimal passageways were cut from the full-term fetuses. These blocks, which encompassed the nasal cavity and lacrimal ducts, were decalcified, trimmed, cleared in oil, and studied with the dissecting microscope to be sure that the ink-stained lacrimal passages were close to the area to be sectioned. After the blocks had been sufficiently trimmed to be sure that the ink-staining duct was about in the center, they were marked with vertical and horizontal pins (Fig. 12*B*). The latter procedure insured the correct plane for sectioning and the obtaining of frontal sections, so that similar areas of the two ducts would be included on the same slide.

Frontal sections were cut 20 to 50 μ thick from the face of the block backward. Only every 10th section was stained until the section containing the nasal end of the duct was located. All the sections immediately before and after this one were used in order that one might have serial sections of the entire lower end of the duct and of its pockets.

This study of the lacrimal ducts in infants showed that the incidence of the imperforate lacrimal duct was much higher in newborn infants than had been expected. Seventy-three per cent of the ducts were not connected with the nose at birth. Two of the infants had one duct open and the other closed. In only two of the infants were both ducts open into the nose. The lower end of the lacrimal duct, although in close apposition to the mucosa of the inferior meatus, was separated from it by a membrane (Figs. 13, 14, 15, and 16). The membrane consisted of the mucosa of the nasal fossa and that of the end of the lacrimal duct, abutting on each other (Figs. 15 and 16). The lower end of the duct was distended and ballooned out under the inferior turbinate (Figs. 14 and 15).

Since this investigation, the nasolacrimal ducts of several other full-term stillborn infants have been studied by distending the lacrimal sac and passageways with saline solution, injected into the lumen of the canaliculus. These, also, have rarely been found to be patent into the nose. To determine when they do become patent, further study of a large number of infants of early age in one of the sizable nurseries should be made.

## SUMMARY

The developmental anatomy of the nasolacrimal duct is reviewed from the earliest appearance of the structure in the embryo to the full-term stillborn infant. The lacrimal passageway arises from an epithelial cord embedded in the mesenchyme, starting at the naso-optic fissure as a thickening of the ectoderm in the 7-mm. embryo. It grows cephalad and caudad to reach the ocular and nasal extremities and canalizes at first in segments; later these coalesce. The ocular end becomes patent, but the nasal end (in less than 30% of specimens) is open only into the nose. An investigation of a number of stillborn infants reveals that in 73.3% the lumen of the lacrimal duct was not connected with the nasal cavity at birth.

527 Sherland Bldg. (1).

Dr. J. Parsons Schaeffer, Professor of Anatomy, Jefferson Medical College of Philadelphia; Dr. L. B. Arey, Professor of Anatomy, Northwestern University Medical School; Dr. Otto Kampmier, Professor of Anatomy, University of Illinois College of Medicine; Dr. George W. Corner, Department of Embryology, Carnegie Institution of Washington, and Dr. A. S. Giordano, South Bend Medical Foundation, gave assistance and cooperation in the preparation of the paper.

# ATYPICAL COLOBOMA OF CHOROID WITH DYSPLASIA OF MACULA

## Report of a Case

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AND
HENRY T. GAYNIN, M.D.
NEW YORK

A CASE of atypical coloboma of the choroid with dysplasia of the macula is presented.

### REPORT OF CASE

A white woman aged 24 consulted one of us because of a minor injury to her left eye. The left eye was markedly divergent, with a deviation of approximately 30 degrees, and vision was reduced to perception of hand movements. Vision was increased to counting fingers at 6 feet (1.8 meters) when the eye was adducted.

Retinoscopic studies of the left eye revealed the presence of compound hypermetropic astigmatism ($+0.75 \bigcirc +1.00$ cyl., axis 165), but no improvement was obtained with lenses. The lids and lacrimal apparatus were normal, and the palpebral fissure was equal to that of the right eye. Gross inspection and slit-lamp studies of the anterior segment revealed no abnormality other than some stellate pigmentation of the anterior capsule of the lens; the vitreous was of normal transparency and structure.

*Fundus of Left Eye* (Figs. 1 and 2).—The nerve head (*a*, Fig. 2) was represented by a kidney-shaped structure, rather pale, and somewhat elevated, with a "moth-eaten" nasal margin. At the pelvis of this kidney-shaped structure, which would ordinarily correspond to the physiological cupping of a normal disk, retinal vessels emerged in atypical fashion. The central retinal artery bifurcated intraneurally, and the secondary branches emerged in a dispersed fashion. There arose from this site a cylindrical band of semitranslucent tissue (*f*, Fig. 2), which flared out temporally and slightly downward to form a large raised mound of semitranslucent tissue, about 2.5 disk diameters in extent and elevated about 4 D. (*c*, Fig. 2). This mound of tissue was capped by an irregularly pear-shaped, opaque-white mass (*e*, Fig. 2).

The mound of tissue descended abruptly to the retinal level on its temporal and inferior aspect, but superiorly it formed an arcuate veil, which swept upward to terminate at the margin of a sharply demarcated circular retinal defect, the size of an average optic disk. This defect (*b*, Fig. 2) formed a sharply demarcated circle with punched-out margins, the base of which was white and ectatic.

Irregular and variably sized clumps of pigment were seen at the margins at 11 to 12 o'clock and at 5 o'clock. Within the circular area, at 8 o'clock and between 3 and 4 o'clock, were two small pigment clumps (Fig. 1). The marginal area within the circle formed a pinkish vascular periphery, whereas the center was pearly white and resembled the floor of a typical coloboma. The central part of the floor was on a lower level than the periphery. This difference was demonstrated by a superior temporal retinal artery which coursed up from the malformed disk to enter the circular defect just slightly temporal to the center and which dipped down to the floor to emerge again at the upper margin between 11 and 12 o'clock. At 10 o'clock, however, a curious vascular development was present, in that an arterial loop seemed to emerge from the floor of the circular defect and send a branch to anastomose with the superior retinal arteries, while other branches coursed upward over the retina.

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Assistant Professor of Ophthalmology, New York Medical College (Dr. Saltzman); Chief of Eye Clinics, Department of Welfare, City of New York (Dr. Gaynin).

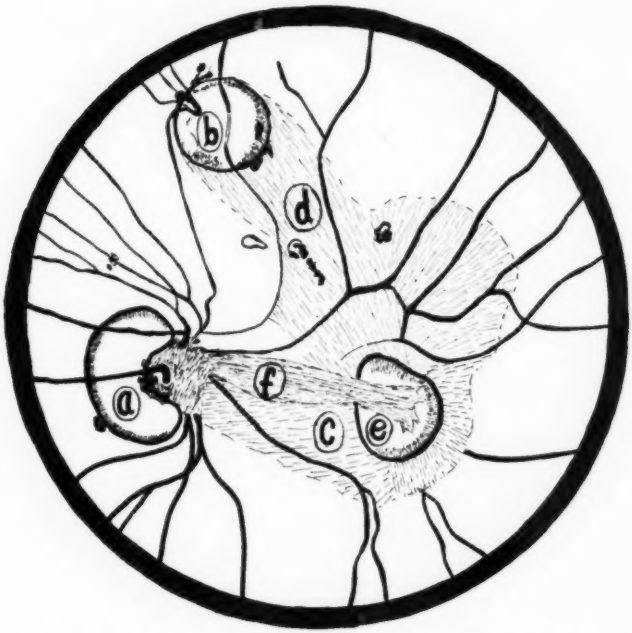


Fig. 2.—Drawing of the fundus: *a*, nerve head; *b*, defect in the retina; *c*, mound of semitransparent tissue; *d*, arcuate veil; *e*, pear-shaped tissue capping the mound; *f*, band of tissue; *g*, Bergmeister's papilla.

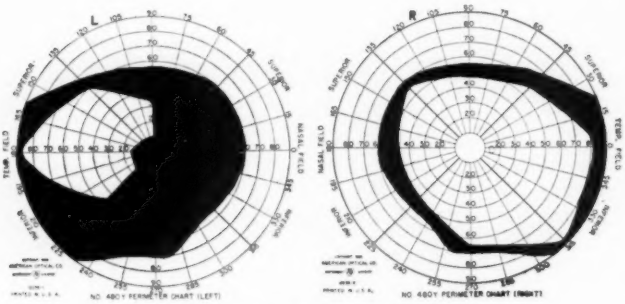


Fig. 3.—Visual fields for 5/330 white.

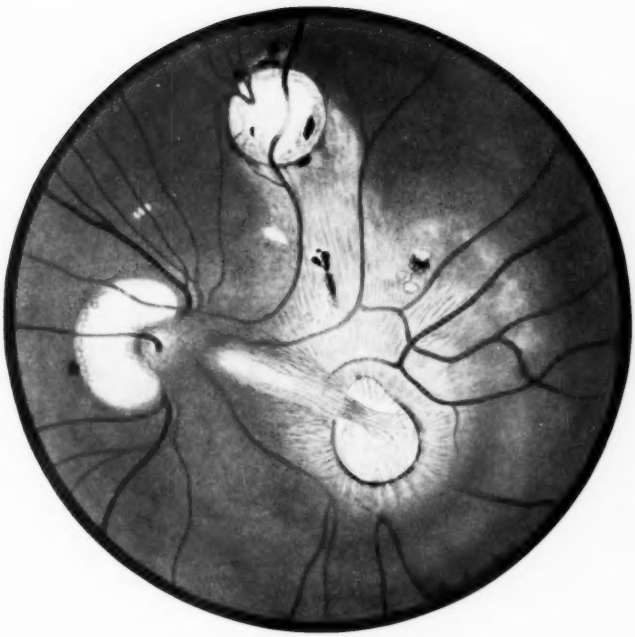

Fig. 1.—Fundus of the left eye.

The retinal mound previously referred to occupied the region of what would normally have been the macula. It was a raised, semitranslucent structure, which rose rather abruptly on its temporal margin but extended as a ridge toward the pelvis of the kidney-shaped disk. The apex of the mound was flattened and capped by the pear-shaped, opaque-white structure, previously described (*e*, Fig. 2). From the center of this opacity, a thick strand of white opaque fibers traversed the top of the ridge to terminate at the temporal side of the malformed disk. The superior margin of the mound, in its central portion, sent forth an arcuate veil (*d*, Fig. 2), which was thin and transparent and extended to the temporal and inferior edge of the circular defect from 3 to 7 o'clock and appeared to terminate there. There were several white patches scattered throughout the fundus, two of which were associated with small pigment masses.

The right eye was normal in every respect. Vision was 20/20 (Snellen), and there was no refractive error.

*History.*—The patient stated that she had noticed poor vision in her left eye at 12 years of age, when she was examined because her "left eye had turned out." She was told to wear a patch over the eye but did not follow this advice. No further medical advice concerning her left eye was sought until she met with the minor injury, as stated above. She had "double pneumonia" at the age of 2 years, and chorea between the age of 10 and 14, during which time she had several "attacks"; these have not recurred since. At 13 years of age, a growth was removed from her left nostril. The gingivae became diseased at the age of 18, and extensive dental extractions were done. Examination of the eyes of the patient's mother, sister, and son revealed no abnormalities. A brother had "a turning in of the left eye" in childhood, but this condition "straightened itself out."

*Laboratory Studies.*—The findings were not significant.

*Field Studies.*—The visual fields are shown in Figure 3.

*X-Ray Studies.*—The x-ray studies of the orbital regions revealed the left superior orbital fissure to be somewhat larger than the right. No clinical significance was attached to this finding, since this fissure frequently varies in size. Roentgenograms of the skull revealed no abnormalities.

## COMMENT

We have assumed that this unusual fundus picture can best be explained on the basis of a prenatal malformation, resulting from fetal inflammation.

In order to explain the origin of developmental defects, one must distinguish between aberration of growth and those abnormalities which may arise as a result of fetal disease.[1] Typical congenital coloboma occurs most frequently at the region of the fetal cleft.[2] Mann [3] called this the fluid, or unstable, region because the possibilities of malformations and atavistic influences are increased in this area. When noxious agents affect the fetal tissues very early, before differentiation takes place, a retardation or perversion of growth results, so that deformities are produced,[4] whereas the same agents affecting the fetal tissue later in development produce a reaction somewhat similar to that in the adult, viz., fibrosis, repair, and alteration of tissues, as end-results of inflammation.[5]

In the differential diagnosis several factors were considered to explain the presence of the semitranslucent tissues, viz., persistent hyaloid artery, retinitis proliferans, and congenital retinal folds.

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1. Mann, I.: Developmental Abnormalities of the Eye, London, Cambridge University Press, 1937, p. 10.
2. Mann,[1] p. 81.
3. Mann,[1] p. 14.
4. (*a*) Mann,[1] p. 44. (*b*) Duke-Elder, W. S.: Text-Book of Ophthalmology, Vol. 2, St. Louis, C. V. Mosby Company, 1944, p. 1240. (*c*) Mann, I.: On Certain Abnormal Conditions of the Macular Region Usually Classed as Colobomata, Brit. J. Ophth. **11**:99-115, 1927.
5. Mann,[1] p. 16.

In the case under discussion, we assume that the pathological aberrations arose as a result of a toxic agent occurring late in development. This resulted not only in a fibrotic alteration of tissue but also in the failure of embryonic tissue to atrophy and disappear. The persistence of Bergmeister's papilla at the pelvis of the malformed disk (*g*, Fig. 2) is, we believe, evidence of such failure of atrophy to occur. The cells of the papilla, which are glial, proliferate during the fifth month of fetal life to form the glial sheath of the hyaloid artery. These cells originate from that part of the inner wall of the optic cup which surrounds the upper end of the fetal fissure.[6] The exact amount of atrophy determines the depression in the center of the disk left by the absorption of the cone cells.[6] Excessive development of Bergmeister's papilla, however, may lead to the formation of strands of whitish tissue, which veil the vessels on the disk and may spread along the retinal vessels.[7] Botteri [8] stated that this tissue may be so dense as to obscure the retinal vessels. The veils may be ectodermal or mesodermal in origin. Rarely, a more solid or sharply defined mass persists, the strands of which may vary from translucent to dense white.[9] These tissues are known as the prepapillary or epipapillary membrane.

Van Manen [10] cited a case of congenital anomaly of the fundus in which he considered that hyperplasia of Bergmeister's papilla was present. He thought the cause of the anomaly was a fetal adhesion between the wall of the peripheral hyaloid vessel and the inner layer of the retina. Samuels [11] stated that to be considered congenital strands at the nerve head must be traceable to the nasal side in the horizontal meridian. All delicate, transparent veils on the nasal side of the nerve are of pure glial origin and should be referred to as neuroglial membranes. He stated that they are never on the temporal side of the papilla. The usual strands and membranes following inflammation have no connection with any remnants of the hyaloid artery. He also stated that unless tissues in diseased eyes accurately fulfil the typical requirements of congenital tissues as to location, extent, and shape they are likely to be of acquired origin. De Beck [12] classified the anomalies which arise through failure of absorption of the embryonic hyaloid artery into 12 groups: (1) shreds of tissue on the optic nerve; (2) membrane on the disk; (3) cystic remains on the disk; (4) masses of connective tissue on the disk; (5) rudimentary strand attached to the disk; (6) strand attached to the disk with vestige also at the posterior surface of the lens; (7) strand passing from disk to lens; (8) similar strand containing blood (subgroup); (9) strand attached to the lens alone; (10) posterior capsular cataract; (11) striae on the posterior lens capsule; (12) persistent canal (without remnants of the vessel).

6. Mann,[1] p. 92.

7. Mann,[1] p. 130.

8. Botteri, A.: Beitrag zu den seltenen angebornen Augenhintergrunds-Befunden, Klin. Monatsbl. Augenh. **48**:11-16, 1910.

9. Mann,[1] p. 132.

10. Van Manen, J. G.: A Congenital Anomaly of the Fundus Oculi, Arch. Ophth. **26**:1-5 (July) 1941.

11. Samuels, B.: Epipapillary Tissues, Tr. Am. Ophth. Soc. **29**:242-252, 1931.

12. De Beck, D.: A Monograph of the Congenital Anomalies and Hereditary Diseases of the Eye, Am. Ophth. Monog. **3**:1, 1890.

When a prepapillary membrane spreads out as a dense mass over a considerable portion of the fundus, it becomes difficult to differentiate it from an inflammatory or a hemorrhagic sequel, or even from a pseudoglioma.[13] Bedell[14] stated that an epipapillary membrane is often confused with inflammatory tissue.

Retinitis proliferans, according to Duke-Elder,[15] is a purely descriptive term which is applicable to several pathological conditions, all of which have a common feature, that of proliferating masses of fibrous tissue in the vitreous following hemorrhage into the vitreous body. The tissue may assume any shape, from broad flat surfaces to compact bands. Klien[16] classified retinitis proliferans histologically into two types. The membrane of Type 1 is dense, opaque, and white or grayish-white. The primary cause of this type is inflammatory or traumatic alteration of the retinal vessels. The membrane of Type 2 has in the earlier stages a delicate network of fibroblasts and proliferating neuroglial tissue within the strands. The primary cause of this type is degenerative vascular disease, leading to circulatory thrombosis and venous anastomosis.

Knapp[17] described a condition in which vitreous opacities accompanying acute exudative choroiditis do not absorb but form a band of opacity, which extends from the nerve head to the area of choroidal atrophy. He described an unusual change in which the vitreous opacity is situated just in front of the retina and seems to have a predilection for the macular area. According to Knapp, these opacities may be differentiated from retinitis proliferans. Loeb[18] discussed a case in which numerous blood vessels were described as though they were in a "transparent membrane lying out in the vitreous." He drew no definite conclusion from this case, believing that the condition was either a congenital anomaly or productive retinitis.

Finnoff[19] described a case in which a veil-like membrane had its origin on, and covered the upper third of, the disk. The distribution of the retinal vessels was anomalous. He thought that the condition in his case was a congenital anomaly rather than retinitis proliferans.

Persistent hyaloid artery may assume a variety of forms. It may appear as large membranes or fibers, showing little resemblance to vascular remnants.[20] The whole vessel may persist and pass across the vitreous to be attached to the posterior pole of the lens, or only the anterior or posterior end may persist. Sometimes the

13. Duke-Elder,[4b] p. 1378.

14. Bedell, A. J.: Some Anomalies of the Fundus: Stereoscopic Photographic Demonstrations, J. A. M. A. **98**:449-454 (Feb. 6) 1932.

15. Duke-Elder, W. S.: Text-Book of Ophthalmology, Vol. 3, St. Louis, C. V. Mosby Company, 1947, p. 2604.

16. Klien, B.: Retinitis Proliferans: Clinical and Histological Studies, Arch. Ophth. **20**:427-436 (Sept.) 1938.

17. Knapp, A.: Formation of Preretinal Connective Tissue in the Vitreous in Acute Choroiditis: Report of 3 Cases, Arch. Ophth. **18**:558 (Oct.) 1937.

18. Loeb, G.: Congenital Anomaly or Productive Retinitis, Am. J. Ophth. **10**:272 (April) 1927.

19. Finnoff, U. C.: Anomalous Condition of the Ocular Fundus, Am. J. Ophth. **3**:161 (March) 1920.

20. Duke-Elder,[4b] p. 1377.

portion that persists may be very close to the disk and appear as a small, sessile, cyst-like body or may be a fibrous cord projecting into the vitreous and attached at one end to the retina.[21]

Danis [22] described a case of persistent hyaloid artery and canal of Cloquet (central canal of the vitreous). A flask-shaped mass covered the site of the papilla, and along a ribbon-like mass there were foci of choroiditis with strongly pigmented spots. He stated that fetal retinitis may produce an adhesion between the retina and the hyaloid artery and its sheath. He further stated that the canal of Cloquet, epipapillary membranes, and what he described as extensions of the lamina cribrosa were essentially congenital malformations. He expressed the belief that all three conditions are dependent on hyperplasia or nonabsorption of the hyaloid tissue.

A congenital fold is characterized by the appearance in the fundus of a band of grayish-white tissue extending from the disk outward to the periphery. Blood vessels accompany the fold and are often closely intermingled with its substance. As a rule, the fold extends to the posterior surface of the lens, which shows subcapsular cataractous changes in the affected region. The fold is usually bilateral and symmetrically disposed. It is found in any meridian and is not necessarily limited to the region of the fetal fissure.[23] Theodore and Ziporkes [24] noted the poor development of the macula, associated with greatly impaired vision. They also observed that congenital retinal fold superficially resembles retinitis proliferans.

Congenital retinal fold is due to a disturbance in growth of the whole of the inner layer of the optic cup. It is a deformity believed to be produced by an abnormal adhesion of the primary vitreous to the inner layer of the optic cup, which does not separate as the secondary vitreous appears. This results in a pulling-up of the retina in a narrow, tent-like detachment. The abnormality occurs mostly after the 13-mm. stage, when the cleft is closed.[25] In all cases the whole of the retina, in addition to the fold, shows imperfect differentiation.[26] According to Tillema,[27] it is conceivable that slight and transitory inflammation of the inner layer of the optic cup may arrest normal development. Weve [28] pointed out that this congenital anomaly is not inflammatory in origin, since it is frequently associated with typical developmental abnormalities of noninflammatory origin, viz., persistent hyaloid artery, persistent pupillary membrane, and nystagmus. He also called attention to the presence of familial and hereditary factors.

The pear-shaped white mass (*e*, fig. 2) capping the semitranslucent mound (*c*, fig. 2) in our case is related either to a condensation of the fibrous tissue in this area or to ectopic bands of nerve fibers. Before the full differentiation of the retina, a definite nerve-fiber layer forms. Aberrant nerves often form two definite

21. Mann,[1] 141-145.

22. Danis, M.: Congenital Anomalies of the Fundus of the Eye, Am. J. Ophth. **4**:233 (April) 1921.

23. Guerry, D.: Congenital Retinal Folds: Report of 2 Cases, Am. J. Ophth. **27**:1132-1135 (Oct.) pt. 1, 1944.

24. Theodore, F. H., and Ziporkes, J.: Congenital Retinal Folds, Arch. Ophth. **23**:1188-1197 (June) 1940.

25. Mann, I.: Congenital Retinal Fold, Brit. J. Ophth. **19**:641-658, 1935; footnote 1, p. 220.

26. Mann,[1] 227.

27. Tillema, A.: Infantile and Congenital Folds, Brit. J. Ophth. **21**:94-98 (Feb.) 1937.

28. Weve, H.: Ablatio Falciformis Congenita (Congenital Retinal Fold), Brit. J. Ophth. **22**:456-470, 1938.

bands running up on either side of the still open cleft toward the optic stalk. This they do not join but form, instead, an abortive second optic nerve. Such small patches of fibers may occur practically anywhere in the wall, on the optic vesicle, or on the optic stalk.[29]

Chorioretinitic lesions have been described in association with dysplasia of the macula; these lesions were assumed to be due to intrauterine inflammation.[30] A case of heterotopia of the macula has recently been described in which no associated inflammatory lesions were seen.[31] Friedman[32] described a case in which heterotopia of the macula occurred as an unusual sequel to retinal hemorrhage from a birth injury.

We consider the circular retinal defect to be an atypical coloboma not related to faulty closure of the fetal cleft. The typical coloboma is situated in or near any part or the whole of the line of the fetal cleft.[2] A typical coloboma is rarely found above the disk.[14]

Johnson[33] referred to extrapapillary coloboma as one which does not involve the optic disc and which is therefore distinct from the classic coloboma, owing to imperfect closure of the fetal cleft. The sclera is of a dazzling whiteness and is sometimes covered with a layer of connective tissue. The presence of connective tissue in the atypical colobomatous regions is explained by a compensatory attempt to close the defect.[34] The etiologic factor in atypical coloboma is a defect of the pigment epithelium, which prevents proper development of the choriocapillaris.[35] This failure in development, in turn, affects the sclera and the inner retinal layers. Van Duyse[36] believed that developmental ectodermal defects result in atypical coloboma.

Mann considered inflammation to be the cause, whereas Klien[37] believed that no generally acceptable explanation exists.

We believe that the white patches and pigment deposits in our case are foci of choroiditis with migration of pigment toward the inner layers of the retina, rather than a congenital pigmentation of the retina. Mann[38] described a type of so-called congenital pigmentation in which the pigment is diffuse, lies on the surface of the retina, obscures the vessels in many places, and is associated with holes and atrophic patches in the choroid. She described a case in which this condition was found and concluded that it was "obviously . . . an ordinary pathological process (reaction to trauma) coinciding with birth and so simulating a congenital anomaly."

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29. Mann,[1] p. 96.

30. Triebenstein, O.: Über Heterotopie des Sehnerven und der Fovea centralis, Klin. Monatsbl. f. Augenh. **62**:442-455, 1919.

31. Cohen, I. J., and Weisberg, H. K.: Vertical Heterotopia of the Macula, Arch. Ophth. **44**:419-423 (Sept.) 1950.

32. Friedman, B.: An Unusual Disciform Retinal Lesion with Heterotopia of Macula, Arch. Ophth. **28**:444 (Sept.) 1942.

33. Johnson, G. L.: Extrapapillary Colobomata, Arch. Ophth. **19**:1-29, 1890.

34. Rones, B.: Genesis of Atypical Ocular Coloboma, Am. J. Ophth. **17**:883-889, 1934.

35. Lloyd, R. I.: Embryologic Basis of Extrapapillary Coloboma and Cornus, Tr. Am. Acad. Ophth. **47**:7-16 (Sept.-Oct.) 1942. Mann,[1] p. 98.

36. van Duyse, M.: Pathogenie des colobomes chorioretiniens atypiques (centraux, paracentraux, extrapapillarires), Arch. d' opht. **37**:22-34, 1920.

37. Klien, B.: Abnormalities of the Posterior Segment of the Human Eye: Embryological Interpretation, Tr. Sect. Ophth., A. M. A., pp. 143-163, 1936.

38. Mann,[1] p. 154.

We consider the abnormal distribution of the retinal vessels in our case to be a congenital anomaly. Finnoff [19] described a case of a congenitally anomalous distribution of vessels; that is, there was no apparent distribution of retinal vessels in the upper nasal quadrant. Mann [39] reported a case of atypical coloboma associated with abnormal blood vessels in which the upper temporal quadrant of the retina was supplied by a group of adventitious blood vessels appearing in the fundus through the localized defect in the retina and choriocapillaris above and outside the disk. An anastomosis was found between the "diminutive" superior temporal vein and the choroidal vessels, which were easily seen between the disk and the coloboma, at the nasal edge. She explained "that some abnormal process has destroyed the membrane of Bruch and allowed an anastomosis between retinal and choroidal vessels in a situation where this never occurs normally in mammalian ontogeny or phylogeny."[11]

In the present case, a branch from the arterial loop (an adventitious vessel), which seems to emerge from the floor of the atypical coloboma, courses upward over the retina (*h*, fig. 2; fig. 1 and description of the fundus of the left eye). Although no anastomosis was seen between the retinal and the choroidal vessels, there did exist an anastomosis between a branch of the arterial loop and a superior nasal branch of the central retinal artery.

## SUMMARY AND CONCLUSIONS

We present a case of atypical coloboma of the choroid with dysplasia of the macula, malformation of the optic disk with aberrant retinal vessels and nerve fibers, retention of Bergmeister's papilla, and foci of disseminated choroiditis.

Normal developmental processes may be interrupted or altered by exogenous agents absorbed through the placenta. The pathological processes thus engendered give rise to an imperfect differentiation of cells, leading to dysplasia or degeneration of fetal tissues.

It is our belief that the multiplicity of fundal abnormalities in the present case may be explained on the basis of the occurrence of a toxic agent late in development. This resulted in fibrotic alteration of tissues and in failure of certain embryonic tissues to atrophy and disappear.

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Dr. A. L. Kornzweig assisted in clarifying some of the embryologic problems involved. Dr. S. Clifford Gilbert, resident in ophthalmology in Sea View Hospital, prepared the bibliography.

39. Mann, I., and Ross, J. A.: Case of Atypical Coloboma Associated with Abnormal Blood Vessels, Brit. J. Ophth. **13**:608, 1929. Mann,[1] p. 184.

# TRANSIENT MONOCULAR BLINDNESS ASSOCIATED WITH HEMIPLEGIA

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## INTRODUCTION

TEMPORARY obscuration of vision, unilateral or bilateral is not uncommon, especially if under that heading one includes the transient amaurosis associated with spasm of accommodation, glaucoma, and hysteria. The visual disturbance accompanying migraine is another common example. Rarer causes of intermittent blindness include eclampsia, lead poisoning, ergot poisoning, malaria, quinine and tobacco intoxication, polycythaemia rubra vera, and paroxysmal hemoglobinuria. Circulatory deficiency in the territory of the basilar and posterior cerebral arteries may also occasion transient disturbances in vision. The bizarre, rapidly changing field defects complained of in the presence of papilledema, particularly when it is due to intracranial venous thrombosis, are less well known.

In addition to the above types, however, there is a fairly large number of cases of periodic blindness in which the principal derangement is an interruption of the retinal blood flow, usually of one eye only. It is this group of cases which is under discussion in this paper. Of obscure etiology and not lending itself to easy or simple classification, this condition has been the subject of sporadic reports during the past century. Intermittent monocular blindness due to retinal anemia constitutes a large fraction of what has been labeled amaurosis fugax, and it is probably better known under that name. Duke-Elder [1] includes cases of this condition under "angiospasm" and divides them into two classes: cases occurring in young persons, who frequently present other evidence of cardiovascular irritability, and cases occurring in the middle-aged or elderly who have pronounced organic disease of the retinal arteries, and who usually have arteriosclerosis or syphilis. This view is perhaps an oversimplification.

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1. Duke-Elder, W. S.: Text-Book of Ophthalmology, London, Vol. 3, Henry Kimpton, 1940, p. 2566.

A careful survey of the literature of the past century has revealed some 150 cases which fall into the category of intermittent blindness, most of them of the monocular type. A summary of these cases will be presented later, and at this point only a few salient features will be noted. As regards the cause, a large number occurred in elderly people and were attributed to arteriosclerosis. In other cases, migraine, Raynaud's syndrome, heart disease, factors acting reflexly, and arteritis, were blamed. However, the cause in the majority of cases was obscure.

This type of blindness has many constant features, as well as many that seem to be highly variable. Characteristically, the blindness lasts from a few seconds to a few minutes. Blindness is usually complete in the affected eye, although at times the defect is limited to one sector. The frequency of attacks varies from several a day to a few each year. Symptoms may last for years or may disappear

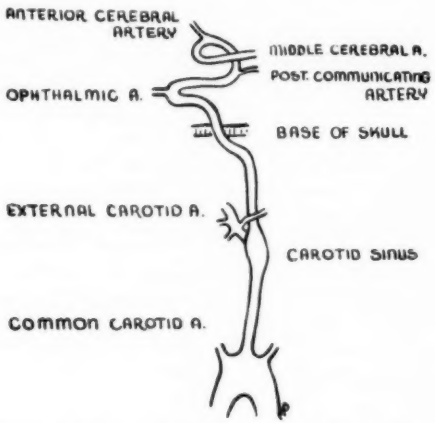


Diagram of lateral view of left carotid system. The atherosclerotic deposit is usually strikingly localized to the carotid sinus. At necropsy, in cases of recent stroke, the internal carotid artery above the sinus is filled with antemortem blood clot.

completely after a few months. In some cases the course of the complaint is benign, whereas in others a few attacks may result in a severe visual deficit.

In recent months I have encountered several cases of transient monocular blindness associated with contralateral hemiplegia, the attacks of blindness usually preceding the stroke, as a sort of warning that disaster threatened. A prominent finding on examination has been occlusion of the internal carotid artery on the side of the affected eye. Attention has not previously been drawn to this clinical syndrome, although transient blindness was described in one of Andrell's [2] cases of thrombosis of the internal carotid artery. My interest was attracted to this subject when within the same week two patients reported transient blindness in one eye as a prodrome of the development of severe hemiplegia. As more attention was paid to patients' complaints about their vision, other cases were encoun-

2. Andrell, P. O.: Thrombosis of the Internal Carotid Artery: A Clinical Study of 9 Cases Diagnosed by Arteriography, Acta med. scandinav. **114**:336, 1943.

tered, and this paper is a report of seven. Disease of the internal carotid artery will no doubt help to explain at least some of the previously reported cases of amaurosis fugax, particularly in the older age groups. More general information concerning occlusion of the internal carotid artery is contained in my communication on that subject.[3]

The seven cases in my personal experience are presented, and a summary of the 150 cases described in the literature follows.

## CLINICAL MATERIAL

The first four cases have been included in a previous study of occlusion of the internal carotid artery.[3]

CASE 1.—*Occlusion of right internal carotid artery within carotid sinus, diagnosed clinically and confirmed post mortem; severe left hemiplegia three years before, preceded by transitory blindness of right eye and attacks of numbness of fingers of the left hand.*

A man aged 68 was found struggling to get out of bed one morning in June, 1947. It was apparent that he had a severe left hemiplegia. He was not unconscious but for several weeks was confused, forgetful, and incontinent of urine. There was very little recovery from the paralysis, and when examined 2½ years later, he could walk only very slowly, using a cane. During the two months before the onset of the hemiplegia, on five or six occasions he had had transient blindness of the right eye, each attack lasting about three minutes. He said, "Wasn't it funny that it came on the wrong side?" He was quite certain he was blind in only one eye and said that it was as though a blind had been pulled down. An ophthalmologist who examined him found nothing remarkable. During the period prior to the stroke he frequently remarked to his wife, "I don't seem to have much feeling in my fingers." He referred to the left hand, and the attack would pass off in a short time. He did not complain of headache. The transient attacks of blindness did not recur after the onset of the cerebrovascular accident.

When he was first examined, 2½ years later, there were severe left hemiparesis and impairment of cortical sensory discrimination on the same side. The visual fields were full; the pupils were equal and reacted to light. Ocular movements were normal, and there was no nystagmus. The fundi were not remarkable. Careful examination of the pulsation in the carotid arteries revealed no difference between the two sides. The blood pressure was 160/90. The arterial pulsations were normal in the lower extremities. The Wassermann reaction of the blood was negative. There was no evidence of diabetes mellitus or anemia.

The patient's final illness was carcinoma of the rectum, for which an abdominoperineal resection was performed on June 16, 1950. After operation circulatory collapse occurred, and he died three hours later.

*Pathologic Findings.*—An extensive cystic focus of old destruction involved the anterior four-fifths of the right frontal lobe, the insula, and the outer portion of the lenticular nucleus. The cerebral arteries showed minimal to moderate atherosclerosis, which at no point seriously narrowed the lumen. The region of the right carotid sinus was unusually firm to palpation. On section, its lumen was almost completely obliterated by yellowish tissue, being reduced to about 1 mm. just above the bifurcation of the common carotid artery. The lumen of the right internal carotid artery above the bifurcation was empty. The other vessels in the neck showed minimal atherosclerosis.

Microscopically, the occluding tissue appeared for the most part to be dense collagenous connective tissue, but in the deeper portion there was atheromatous material. Abundant small, endothelium-lined capillaries were scattered through the atherosclerotic mass.

This case represents the most typical picture in my present experience. Several attacks of blindness had occurred prior to the stroke, but none after it. The eye was not affected at the time of onset of the hemiplegia. The occlusion of

3. Fisher, M.: Occlusion of the Internal Carotid Artery, A. M. A. Arch. Neurol. & Psychiat. **65**:346 (March) 1951.

the carotid artery was in the region of the carotid sinus and was due chiefly to atherosclerosis, although superimposed thrombosis may well have been present at the time of the total occlusion. Pulsation in the internal carotid artery on the affected side did not seem to be diminished. Failure to detect absence of pulsation in this artery was due either to faulty technique or to the examiner's mistaking pulsation in the external carotid artery for that in the internal carotid artery. The ophthalmic artery, the central retinal artery, and the eye were not examined pathologically, as I did not appreciate the uniqueness of the visual symptoms at that time.

CASE 2.—*Occlusion of right internal carotid artery found on surgical exploration for carotid arteriography; history of left hemiplegia, preceded by transient attacks of blindness in right eye and right-sided headache. Right optic atrophy found seven months later.*

A man aged 60 awakened one morning with weakness and numbness of the left hand and forearm, associated with frontal headache. His complaints disappeared in a few hours, and he was well until the following day at noon, when the numbness returned, but cleared within a few hours. The following morning he awakened with complete paralysis of the left side of the face and the left arm and leg.

On closer questioning, it was found that during the month prior to admission he had several times lost vision in the right eye for approximately a minute. On one occasion he said to a friend, "I've gone blind in one eye." His friend replied, "Don't worry; it will be all right in a minute," and it was. The patient, therefore, paid no more attention to the symptom. The blindness was described as "a fog rolling in," and he was certain that only the right eye was involved. The blindness lasted about two minutes, the fog simply rolling away again. For at least two years the patient had had recurrent headaches on the right side, above and to the side of the eye, which were steady in character and lasted a half-hour to an entire day. The headache came about once a month and was not severe.

Examination 24 hours after the onset of the stroke showed total left hemiplegia associated with a sensory deficit and homonymous hemianopsia. The pupils were equal and reacted to light. Ophthalmoscopic examination showed almost normal retinal vessels. There was no impairment of visual acuity. Pulsation was absent in the right internal carotid artery. The blood pressure was 170/90. Sinus (cardiac) rhythm was normal. An electrocardiogram was not remarkable. Surgical exposure of the right carotid vessels for arteriography revealed good pulsation in the common and external carotid arteries but no pulsation in the internal carotid artery, and no blood could be withdrawn from it with a syringe and needle.

Recovery was slow and incomplete, the left hand remaining useless. On reexamination, seven months after the stroke, the right optic disk was white, the retinal arteries were reduced to fine pink threads, and the visual acuity of the right eye was reduced to reading the largest headlines. The patient did not realize that vision was so impaired and could not recall the time of onset of the blindness. The left eye was normal.

This case, again, exemplifies several features associated with occlusion of the internal carotid artery, namely, prodomal transient blindness, headache, and a slow, "stuttering" onset of the hemiparesis. It is unusual for optic nerve atrophy to appear later, this being the only instance in the present series. The retinal picture is similar to that of thrombosis of the central retinal artery. The diagnosis of occlusion of the internal carotid artery was confirmed.

CASE 3.—*History of transient blindness preceding and following right hemiplegia of gradual onset; absence of pulsation in internal carotid artery.*

A man aged 66 arose one morning at 5:45 a. m. One-half hour later, while he was putting on his shoes, his right hand became paralyzed, and the medial side of the hand and forearm felt as though he had "bumped his funny bone." He was alone and did not have occasion to speak. Fifteen minutes after the onset of the weakness, the hand was stronger, and he was able to use it in taking tram tickets from his pocket. At 6:40 a. m. he spoke to the tram conductor.

Within a few minutes his hand became weak again, and he was unable to say a word to a friend. Ten minutes later he could speak normally, but the hand remained weak. Aphasia returned for 20 minutes or so at 9 a. m., and at 10 a. m. it came on again and persisted until his admission to the hospital, a few hours later. He did not have a headache.

For three or four months prior to admission, transient attacks of blindness of the left eye had occurred repeatedly. They lasted from one to seven minutes and had gradually increased in frequency to a maximum of two or three a week. If he closed his good eye during an attack, he was totally blind. The blindness came on as though a screen were raised diagonally from the lower medial portion of the upper outer border of the visual field. Return of vision was in reverse, and vision in the upper outer field might be perfect while the blackness was retreating below. Vision was never colored during the period of recovery. The onset was gradual, as was the recession, each being of about the same duration as the period of total blindness. He also had several episodes of numbness of the medial one and one-half fingers of the right hand and of the forearm, but these never came on at the same time as the blindness.

When admitted, he was aphasic, and the right side of the face and the right hand were weak. There was a Babinski response on the same side. The pupils were equal and reacted to light and in accommodation. There was no visual-field defect. The fundi were not remarkable. Pulsation in the left internal carotid artery was absent. The blood pressure was 180/82. Normal sinus rhythm was present. The Wassermann reaction of the blood was negative.

Recovery from the paralysis was rapid, but slight difficulty in finding the appropriate word was present even 12 months later. In the interval, transient blindness has continued to occur about four times a week. The eye could be examined ophthalmoscopically near the end of one attack, but the only change noted was that the arteries, which were at first greatly narrowed, distended fully within about 30 seconds. No pallor was present. The blindness was not associated with any subjective sensation, such as dizziness or lightheadedness. A diary was kept in an attempt to find factors related to the onset of the attacks, but the episodes seemed to occur haphazardly, not being related to exercise, sleep, reading, eating, or position. Arteriography has been avoided because of the precarious state of the circulation in the left eye. Compression of the left common carotid artery for 15 seconds did not cause an attack of blindness.

This case is only presumed to be one of occlusion of the internal carotid artery, but the typical clinical picture, combined with the absence of pulsation in the left internal carotid artery, is considered good evidence in favor of that diagnosis. Persistence of transient blindness after the onset of the stroke is unusual. It would have been most useful to know whether or not the internal carotid artery was entirely occluded, for the site of the intermittent blockage could then have been sought elsewhere than in that artery; but arteriography has been avoided. The "march" of blindness across the visual field from below and medially to above and laterally should prove to be a common finding, if it is true that the superior temporal branches of the central retinal artery are most prone to involvement in ischemia of the retina. The fact that blindness never occurred at the same time as the other transient symptoms is characteristic. The failure of compression of the common carotid artery to bring on blindness will be considered later.

CASE 4.—*Severe left hemiplegia, preceded by intermittent blindness in the right eye for seven months; transient cerebrovascular stroke 25 years before; absence of pulsation in right internal carotid artery.*

A man aged 49 one day while at work noticed numbness of the medial two fingers of the left hand. It gradually ascended the arm, and the hand became weak and useless. Two hours later the left leg became numb and weak. He was able to walk a short distance to his home. Next morning, on his awakening, the left arm and leg were almost useless. Careful inquiry then elicited the fact that for at least seven months he had been having attacks of total blindness in the right eye, lasting only a few seconds and occurring once a week. Vision would suddenly blur and fade out, but on his rubbing the eye several times, sight promptly returned. For one week previously he had had headaches over the right eye, each attack lasting one to two hours.

The headache was present on arising and made him feel unwell, but seemed to pass off in the fresh air. At least twice in the week prior to the stroke he had a "pins-and-needles" sensation in the medial two fingers of the left hand, exactly similar to the first symptoms of his later stroke. The blindness did not occur after the onset of the hemiplegia.

When he was first examined, several days after the onset of his illness, there was weakness of the entire left side, the hand being useless. Cortical sensory deficiency was present, but there was no hemianopsia. The fundi were not remarkable. Pulsation in the right internal carotid artery was absent. A normal sinus rhythm was present. The blood pressure was 130/80. The peripheral pulsations in the limbs were normal.

This case, again, is unverified but illustrates the association of transient blindness and severe hemiplegia. The attack of blindness ceased with the onset of the paralysis.

CASE 5.—*Severe hemiplegia 15 years before death; repeated attacks of blindness for one month prior to stroke; carotid arteries not examined at autopsy.*

A man aged 40 was sitting quietly at 11 o'clock one morning in 1935. He leaned over to pick up a newspaper and found that his right hand would not function. His right leg was also paralyzed, and he could not speak. Within two minutes he had recovered perfectly. At 3 p. m. the process repeated itself, this time lasting three minutes. At 5 p. m. he became paralyzed again, and, after partially recovering, he became totally hemiplegic at 7 p. m.

When first seen in 1950, he stated with certainty that for one month prior to the stroke he repeatedly lost vision in the left eye. He was also certain that as a rule the visual loss came only in the left eye, but that occasionally both eyes were affected. The attacks occurred once or twice a day, lasting about half a minute, and it seemed as though a blind were lowered. In 1950, he was still severely paretic, but speech was normal. More recently, his left side had been weak, and on several occasions he had had attacks of vertigo associated with tinnitus, diplopia, and midline occipital headache. Blindness did not recur after the onset of hemiplegia.

Examination revealed spastic paresis of the right side and decreased sensation to pinprick. No pulsation was felt in the left internal carotid artery. The blood pressure was 118/80.

The patient died of hepatic carcinoma on Dec. 25, 1950. Unfortunately, the carotid vessels could not be examined.

The brain was small, and the vessels were almost free of atherosclerosis. The circle of Willis had a large anterior communicating artery, but both posterior communicating arteries were very narrow. The carotid trunks were both large. Upon section, a partially trabeculated cyst (7 mm. in width, 15 mm. in length, and 25 to 30 mm. in vertical diameter) was observed to occupy the posterior and upper two-thirds of the lenticular nucleus and adjacent posterior limb of the internal capsule. The walls of the cyst were not darkly stained.

This case, again, was typical clinically. The old softening within the brain was of an unusual nature and size. Its position was also atypical. No important deductions can be drawn from this case because the carotid vessels in the neck were not examined.

CASE 6.—*Indefinite history of left hemiplegia in 1943; two attacks of blindness in right eye in 1950.*

The patient's memory and intelligence precluded an accurate history. He stated that in 1943 his left side became weak but recovered, with only a mild residuum. He was seen in 1950 because of visual trouble, when he stated he was certain that he had lost vision completely in the left eye twice in the preceding year, each attack lasting five minutes, and that the other eye sometimes blurred. He had had angina pectoris since 1941.

Examination showed traces of weakness of the left side. No pulsation was felt in the right internal carotid or temporal artery. The blood pressure was 115/95 in the right arm and 160/98 in the left arm, and the pulse at the right wrist was faint. No pulsations were felt in the lower extremities except in the right femoral artery. A striking bruit was heard in the right lower quadrant of the abdomen. The fundi were remarkably normal.

This case, because of the unreliability of the information, does not merit special attention. However, it illustrates a not uncommon finding associated with occlusion of the internal carotid artery, that is, evidence of widespread severe atherosclerosis. In this case, the atherosclerosis seriously affected the lower limbs, iliac arteries, aorta, coronary arteries, and right subclavian and common carotid arteries. Transient attacks of blindness had appeared several years after the patient's stroke. I have seen another such case, not included in this paper.

The following case is included because of its similarity to the first six cases, but the pathologic basis is probably much different, and for that reason general statements made throughout the paper refer primarily to the other cases.

CASE 7.—*Recurrent transient field defect in left eye for 5 to 10 years; recent stroke, with moderate recovery; high blood pressure.*

A woman aged 46 was sewing one day when her right hand became weak and numb. She could no longer use it for sewing. At the same time mild dysphasia appeared. Recovery began in two days, and while the right hand was soon normal again, there was still difficulty in naming objects six weeks later. One month before the stroke there had been an attack of difficulty in speaking for five minutes.

She also stated that for 5 or 10 years she had repeatedly lost vision in the lower field of the left eye. The attacks lasted about three minutes and recurred usually once a week, but not infrequently two or three times a week. Occasionally vision was lost completely in that eye. The blindness came on gradually and passed diagonally across the eye like a screen and receded in the reverse direction. As the attack passed off, the patient would see a changing mosaic of sparks and flashes. Vision was never blue or fiery red. She was sure the phenomenon occurred in one eye, that the lower field of vision was lost, and that there were no associated symptoms. A recent attack had lasted almost an hour.

Examination, six weeks after the stroke, showed that the pupils were equal and reacted to light. The fundi were normal except for narrowing of the retinal arteries. The visual fields were full. There was no trace of the weakness of the right side, and the reflexes were equal on the two sides. When she was first examined, pulsation in the carotid arteries was good, that in the right being more forceful than that in the left; but eight months later pulsation on the left side was almost absent. The blood pressure was 190/110. The Wassermann reaction of the blood was negative.

This case differed in several ways from the previous ones. The transient attacks of blindness involved only the lower visual field of one eye. The tendency of the upper branches of the central retinal artery to evince the effects of ischemia more readily than the lower branches has been referred to in Case 3. Moreover, the patient had moderately severe hypertension, and the carotid pulses were almost normal when she was first examined. It is quite possible that the patient has disease of the ophthalmic or central retinal arteries and that more recently there has developed an unrelated focus of hypertensive encephalomalacia, which caused the weakness of the right side and the aphasia. In other words, this case is not a definite instance of occlusion of the internal carotid artery, but is presented to illustrate the fact that transient visual disturbances associated with a cerebrovascular accident may have more than one cause.

## HISTORICAL SUMMARY

A search of the literature has revealed in the older age group approximately 30 cases of transient monocular blindness which fall in the same general category as the cases just presented. However, only in Andrell's case were there described an associated occlusion of the internal carotid artery and contralateral hemiplegia.

In a few others, hemiplegia alone was described. Since there is no comprehensive summary of all the cases of transient ischemic blindness reported in the literature, it has been anticipated that readers will find it difficult to place my cases in their proper relation to those previously reported. With this in mind, there follows a rather detailed account of all previously reported cases, divided into groups, as outlined below. It is not claimed that the survey is exhaustive, but certainly the great majority of cases are included. The earlier reports lack such data as reactions to the Wassermann test and blood pressure levels, but they more than compensate in the excellence of their exact and detailed clinical descriptions. Sometimes the account is suggestive of an intracranial tumor, a "ridge" meningioma, or a berry aneurysm. However, compression of the optic nerve by such lesions has never been described as intermittent, although there is no good reason that it should not occasionally be so.

The origin of the term "amaurosis fugax" has not been found. The early English ophthalmologists were aware of the condition, for Middlemore [4] (1835) referred to it as "periodical amaurosis." With the perfection of the ophthalmoscope and the introduction of Virchow's doctrine of embolism, it was not long before sudden loss of vision, even if preceded by transient warning attacks, was invariably attributed to embolism of the central retinal artery. Several ophthalmologists, including Loring,[5] Nettleship [6] and Smith,[7] objected to the concept of multiple embolism as the cause of intermittent amaurosis, favoring that of failure of the general circulation. Wagenmann [8] (1897) considered at great length all the possible causes and strongly favored vasospasm as the mechanism. The subsequent history of amaurosis fugax consists of a series of case reports, in which migraine, Raynaud's disease, heart disease, reflex irritation, arteritis, arteriosclerosis, malaria, syphilis, and other conditions have been held responsible. Some 30 observers have examined the fundus during a transient attack of blindness.

The cases described in the following historical summary are divided into seven groups: (1) cases in the older age group, in which atherosclerosis is probably a factor; (2) cases in the younger age group, with no apparent cause; (3) cases associated with migraine; (4) cases associated with Raynaud's syndrome; (5) cases associated with heart disease; (6) cases of reflex amaurosis, and (7) miscellaneous cases. The placing of a case is often arbitrary because of lack of clinical details, but most of the highly atypical cases have been listed in the miscellaneous group. A few cases of bilateral transient blindness have been included. A pathologic examination has so far never been made, and this, with the lack of follow-up studies, constitutes the chief deficiency of the reported cases.

Because of the length of the historical summary, it is recommended that readers not specifically interested in the problem proceed immediately to the section on the clinical picture.

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4. Middlemore, R.: A Treatise on the Diseases of the Eye and Its Appendages, London, Longmans & Co., 1835, p. 303.

5. Loring, E. G.: Remarks on Embolism, Am. J. M. Sc. **67**:313, 1874.

6. Nettleship, E.: Repeated Paroxysmal Failure of Sight, in Connection with Heart Disease, Brit. M. J. **1**:889, 1879.

7. Smith, P.: Reflex Amblyopia and Thrombosis of the Retinal Artery, Ophth. Rev. **3**:1, 1884.

8. Wagenmann, A.: Beitrag zur Kenntniss der Circulationsstörungen in der Netzhautgefässen, von Graefes Arch. Ophth. **44**:219, 1897.

GROUP 1.—*Cases in the older age group, in which atherosclerosis is probably a factor.*

Hughlings Jackson[9] (1865) reported the case of a middle-aged woman who lost all vision for five minutes. There were no spots, specks, or clouds. The visual loss was not due to failure of accommodation, as she stated that it was not like looking through a strong convex lens. Jackson attributed the attack to temporary contraction of the retinal vessels and used the term "retinal epilepsy."

Knapp's[10] (1868) patient, aged 46, had had transient attacks of numbness and weakness of the left side, preceding permanent weakness of that side. Blindness in the right eye occurred one night, but the patient said that transient blindness had occurred on that side once previously. The retinal arteries were thread-like, and Knapp concluded that embolism had taken place. He tried to find the source of the embolism, and since the heart was normal, he auscultated over the carotid vessels and found at the bifurcation of the right common carotid artery a clear murmur, similar to that heard over an aneurysm. Later it was discovered that the carotid vessels did not pulsate on the left side. The similarity of this case to those reported in this paper is most striking.

Smith[7] described the case of a woman aged 40 who after a bout of coughing lost the sight of her left eye. She rubbed her eyelids, and vision returned almost immediately. A few minutes later the eye "went dark" again, and after she rubbed her lids sight came back as before. The next day, while she was walking in the street, the eye suddenly "went dark" again and remained blind. The retinal vessels were extremely fine, and the disk was white.

Wagenmann[8] described the case of a man aged 69 who had had repeated transient attacks of blindness in the right eye, coming on suddenly and beginning at the side or below. The blindness was total; the pupil did not react to light, and ophthalmoscopic examination showed the arteries to be reduced to fine yellow threads, in which no blood could be seen. The arteries could be followed only a short distance from the nerve head and then disappeared. No external changes in the eye took place. Attacks recurred with variable frequency from once in three or four days to several times a day. They lasted up to 10 minutes, and when vision returned everything appeared distinctly blue. Attacks occurred for two months and then ceased after an iridectomy was performed for decompression. At the height of the attack in which the patient was examined, pressure on the eye did not lead to pulsation in the retinal artery. The vein at the nerve head pulsated slightly. After a period of eight months of freedom from attacks, the patient became blind in the affected eye over a period of two days, and the retinal picture was that of thrombosis of the central retinal artery. After a detailed consideration of the possible causes, the author inclined strongly to the view that arterial spasm was responsible, arteriosclerotic changes no doubt predisposing to spastic phenomena.

Sachs[11] (1900) described the case of a man aged 54 who, after having several attacks of blindness in the left eye, awakened to find vision gone permanently. On ophthalmoscopic examination, the inferior retinal artery showed constrictions, which came and went and moved along the vessel in peristaltic fashion. Sachs favored vasospasm as the responsible mechanism.

Posey[12] cited the case of an elderly man who had had three attacks of total blindness in the right eye in two months. The attacks lasted 10 minutes, 15 minutes, and off and on for 5 or 6 hours, respectively. No attacks had occurred in the following two years.

Greenwood[13] (1905) described the case of a man aged 50 who for about a year had been having frequent sudden attacks of blurred vision in the right eye, lasting from a few minutes to an hour. In the last few weeks sight had become poorer in that eye, and he had noticed loss of the upper half of the field of vision (confirmed by perimetry). The patient looked and acted

9. Jackson, J. H.: Observations on Defects of Sight in Brain Disease, and Ophthalmoscopic Examination During Sleep, Royal London Ophth. Hosp. Rep. **4**:10, 1863-1865.

10. Knapp, H.: Über Verstopfung der Blutgefässe des Auges, von Graefes Arch. Ophth. **14**:207, 1868.

11. Sachs, M.: Netzhautarterienkrämpfe in einem unter dem Bilde der Embolie der Centralarterie verlaufenden Falle, Beitr. Augenh. **44**:34, 1900.

12. Posey, W. C.: Transient Monocular Blindness, J. A. M. A. **38**:1418 (May 31) 1902.

13. Greenwood, A.: Obstructions in the Retinal Arteries, J. A. M. A. **44**:781 (March 11) 1905.

like a man enfeebled by age, his memory being poor. On ophthalmoscopic examination, the inferior branches of the retinal artery of the affected eye were found to be reduced to white threads. A striking feature was the presence of several new-formed arterioles running over the disk to the adjacent retina.

Lundie's [14] patient, a man aged 88, suddenly became totally blind in the left eye but recovered vision within an hour. Indirect ophthalmoscopic examination during the later part of the attack, when some vision had returned in the upper field, showed local interruption of the column of blood in the upper main branch of the retinal artery. The artery in a length somewhat less than 1 disk diameter was reduced to a whitish streak. Later, a fine thread of blood could be seen, and when vision was restored the artery appeared normal. There were no further attacks.

Zentmayer [15] (1907) described the case of a woman aged 43 who for 18 months had been having attacks of dizziness, accompanied with temporary blindness. The blindness affected both eyes and lasted from 5 to 10 minutes. At first both eyes recovered fully, but later the lower half of the right visual field was slow to recover. Later this delay became more pronounced, and finally vision was entirely lost from the right lower visual field. The left visual field went through the same process, but the upper half of the field became impaired. The visual disturbances occurred at irregular intervals, but seemed more frequent during the menstrual period. On examination, the upper branches of the central retinal artery of the right eye were found to be contracted and the upper portion of the disk decidedly atrophic. In the left eye the lower portion of the disk was pale, and the arteries were slightly reduced in size. Later she reported "spells of swimming of the sight," lasting from three to five minutes and involving the whole field. She had had a transient sector defect of almost total darkness, extending from fixation upward and outward, lasting about 30 minutes and disappearing gradually. The author attributed the phenomenon to pressure on the optic nerve as it lay between the hardened internal carotid artery and the unyielding dural ring, just behind the optic foramen.

Weiss [16] (1912) reported the case of a man aged 54 who had been blinded in the left eye by an accident as a child. For "some time" he had been having attacks of blindness in the right eye, often three a day. An attack came on while he was being examined, and, in succession, the arteries and then the veins became empty, the vessels becoming yellowish-white threads. After about half an hour the vessels filled again—first the atypical cilioretinal arteries, then the veins, and finally the larger arteries. During this period the patient was blind, but vision returned as the vessels filled. At first vision was blue, then red as fire. The patient also complained that the fingers of the left hand would at times "go to sleep," and Weiss thought his patient had unilateral Raynaud disease. This case could well be one of occlusion of the internal carotid artery.

Eigler [17] (1920) described the case of a man aged 42 who, after having attacks of momentary blindness for over two years, suddenly was seized with complete blindness in the left eye. On examination, the upper temporal artery seemed blocked by a white plug. No other details were given.

Jamieson's [18] patient, aged 88, showed complete anemia of the central retinal artery and its branches for two minutes.

McCoy [19] (1925) cited the case of a woman aged 72 who had had several attacks of obscuration of vision in the left eye, lasting for a few minutes. The present attack came on two hours before she was seen by the author, who found vision reduced to perception of hand movements at 6 inches (15 cm.). The retinal arteries were very pale, and several branches were practically white. Amyl nitrite and massage were exhibited, with improvement of the retinal anemia, but vision remained 20/200.

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14. Lundie, R. A.: Transient Blindness Due to Spasm of the Retinal Artery, Ophth. Rev. **25**:129, 1906.

15. Zentmayer, W.: Some Unusual Ocular Manifestations of Arteriosclerosis, J. A. M. A. **48**:750 (March 2) 1907.

16. Weiss, K. E.: Amaurosis fugax durch Krampf der Retinalgefässe, Ber. Versamml. deutsch. ophth. Gesellsch. **28**:205, 1912.

17. Eigler, C. O.: Obstruction of Central Retinal Artery, Am. J. Ophth. **3**:452, 1920.

18. Jamieson, cited by Crisp, W. H.: Spasm of the Retinal Arteries, Am. J. Ophth. **4**:188, 1921.

19. McCoy, L. L.: Spasm of the Central Retinal Artery, Am. J. Ophth. **8**:140, 1925.

Friedenwald [20] (1926) described two cases. The first was that of a man aged 65 who had had three attacks of right-sided blindness in a period of a week but none subsequently. The blindness lasted a few minutes. The second case was that of a man aged 48 who also had angina pectoris. The patient had had three attacks of blurred vision in two years.

Wagener and Gipner [21] (1927) described the case of a woman aged 47, with hypertension, who eight years before had suddenly and permanently lost some of the vision in the left eye. For several months previously she had had "flickering" in the same field every other day or so, the attacks lasting a few minutes. There had been one right-sided attack, lasting four minutes, during which she could not discern the face of a clock. The lumen of the main inferior temporal artery was much narrowed, and its walls were thickened. The lower temporal quadrant of the optic disk was pale. Auricular fibrillation was present.

Carlotti's [22] patient, a man aged 64, had had repeated attacks of blindness in the left eye over a period of two years. Then an episode occurred in the right eye, leaving small, contracted retinal arteries and an extensive field defect. The patient had syphilis.

Mayou [23] (1931) briefly mentioned a man who had had attacks of obscuration of vision with ischemic appearance of the disk and retina, lasting three to four minutes at a time. Autopsy revealed an aneurysm of the subclavian artery of syphilitic origin and syphilitic heart disease. No further details were given. Involvement of the carotid vessels might well have been a factor in this case.

Foerster and Guttman [24] (1933) described two cases. The first was that of a man aged 34 who had had repeated attacks of loss of vision in the right eye. One attack occurred during examination. Vision was lost first in the lower field, when the vessels running to the upper part of the retina became bloodless for about 1½ disk diameters above the disk. Farther out there was blood again. Severe left hemiplegia also appeared. Cervical and periarterial-carotid sympathectomy was performed on the right side, when it was noted that the carotid artery scarcely pulsated. Apparently, no importance was attached to this finding, and the diagnosis of cerebral thromboangiitis obliterans was made. The second case was that of a man, also aged 34, who had had headache, signs of right hemiparesis, slight aphasia, and repeated attacks of blindness in the left eye.

Bailliart and Rollin [25] (1935) described a man aged 59 who for two or three years had had transient attacks of obscuration of the peripheral visual field of the left eye, lasting but a few seconds. One attack left him with a permanent field defect and narrowing of the corresponding retinal artery. Slight progressive diminution of hearing was also present.

Schiff-Wertheimer and Ballard [26] (1935) cited the case of a woman aged 52 who in May and July, 1934, and in August and October, 1935, had suddenly lost vision in one eye (two attacks in each eye). Total blindness lasted 36 to 48 hours, but vision was impaired for two weeks and normal vision returned only after four weeks. The vessels during one attack were attenuated, and the intraocular pressure was extremely low.

Marinesco and Kreindler [27] (1936) described a fascinating case of bilateral occlusion of the carotid artery in which fleeting attacks of blindness had occurred in the left eye. On exam-

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20. Friedenwald, H.: Sudden and Transient Blindness, Am. J. Ophth. **9**:831, 1926.

21. Wagener, H. P., and Gipner, J. F.: Arterial Spasm in Production of Occlusion of Branches of Central Artery of the Retina, Am. J. Ophth. **10**:650, 1927.

22. Carlotti, in discussion of Second Congress of the French Otoneuro-Ophthalmologic Societies, 1928; reported, Rev. d'oto-neuro-opht. **6**:603, 1928.

23. Discussion, Tr. Ophth. Soc. U. Kingdom **51**:122, 1931.

24. Foerster, O., and Guttman, L.: Cerebrale Komplikationem bei Thrombangiitis obliterans, Arch. Psychiat. **100**:506, 1933.

25. Bailliart, P., and Rollin, A.: Spasme des artères rétiniennes, Bull. Soc. opht. Paris **47**:612, 1935.

26. Schiff-Wertheimer, S., and Ballard, M. H.: Oblitérations répétées de l'artère centrale de la rétine, Bull. Soc. opht. Paris **47**:615, 1935.

27. Marinesco, G., and Kreindler, A.: Obliteration progressive et complète des deux carotides primitives; accès épileptiques; considérations sur le rôle des sinus carotidiens dans la pathogénie de l' accès épileptique, Presse méd. **44**:833, 1936.

ination during an attack, the central retinal artery seemed to be in spasm. Autopsy, later, showed total occlusion of all three of the large vessels arising from the aortic arch, due to an extensive plaque of atherosclerosis.

Sédan and Gaëtan[28] (1936) cited two cases. The first, reported by Bailliart, concerned a man aged 48 with repeated attacks of blindness in the right eye and numbness in the left hand. Suddenly, vision failed completely in the right eye but was recovered for two days; then, as a result of an emotional disturbance, blindness returned. The second case was that of a patient aged 67, originally reported by Redslob, who after gradual diminution of vision in the right eye, had two attacks of blindness, the second of which was permanent.

Sanders[29] (1939) described the case of a man aged 60 who had had attacks of complete blindness in the left eye, usually coming on during exertion while the patient was in the bending position, but also occurring during walking, leaning over, turning the head suddenly, and twice in hospital, while he was lying in bed. Attacks lasted from a few minutes to 10 minutes. They were produced voluntarily by exertion, allowing the author to make a detailed examination of the fundus, as well as obtain photographs. During the period of blindness the retinal arteries were reduced to thin white threads, which could be seen only with difficulty. The veins seemed normal; the disk was pale, and the retina was hazy. The actual closure was not seen, as the patient was exercising. On recovery, the vessels filled several seconds before vision returned, and the veins were dilated afterward for 10 to 15 minutes. General examination showed nothing remarkable. During exercise the patient's blood pressure rose to about 180/100, and it was thought that this was the only causative factor discovered. Glyceryl trinitrate placed under the tongue prevented attacks on two occasions, although violent exercise was carried out. Attacks occurred for a period of two weeks and then ceased.

The second case of Lindenberg and Spatz[30] (1939) concerned a man aged 36 (at death) who at the age of 27 first began to have attacks of periodic blindness in the left eye, associated with headaches. The attack was described as follows: "The onset was with severe headache, followed shortly thereafter by flickering before the left eye. There was a slow decrease of vision, until blindness occurred. Vision gradually returned to normal. The attack under observation lasted two minutes, during which the retinal arteries became snow-white. The blood column moved backward in the superior temporal artery. The superior temporal vein, which was narrowed, appeared to be filled with irregular masses. The arteries of the papilla became slowly red, and vision returned." The attacks gradually lasted longer, and finally the typical picture of occlusion of the central retinal artery appeared. The blood pressure was 148/90. At the age of 36 the patient died of a stroke. Pathologic studies showed changes in the cerebral vessels interpreted as thromboangiitis obliterans. The carotid and ophthalmic arteries were not studied. The brain contained many small softenings.

Wagener[31] (1941) reported occlusion of the retinal arteries after transient attacks of blindness.

Andrell[2] presented several cases of thrombosis of the internal carotid artery. His seventh case was most striking, for right hemiplegia was associated with recurrent blindness in the left eye. Transient blindness occurred about four times a month for five months and began only after the onset of the cerebrovascular accident. Arteriograms of the left side showed occlusion of the internal carotid artery at its origin, but the external carotid artery filled well. Arteriograms of the right side resulted in filling of the cerebral arteries of both sides. His second case was that of a man aged 32 who had had intermittent attacks of headache and

28. Sédan, J., and Gaëtan, E. J.: Considérations sur les spasmes de l' artère centrale de la rétine, Ann. ocul. **173**:609, and 865, 1936; **174**:73, 1937.

29. Sanders, T. E.: Intermittent Occlusion of the Central Retinal Artery, Am. J. Ophth. **22**:861, 1939.

30. Lindenberg, R., and Spatz, H.: Über die Thromboendarteritis obliterans der Hirngefässe (cerebrale Form der v. Winniwarter-Buergerschen Krankheit), Arch. path. Anat. **305**:531, 1939.

31. Wagener, H. P.: The Significance of Spasm in Retinal Arteriolar Disease and in Retinitis, Tr. Pacific Coast Ophth. Soc. 1939, 27th meeting, pp. 165-177; abstracted, Am. J. Ophth. **24**:345, 1941.

"glistenings" for four years prior to the onset of severe hemiplegia. Arteriograms showed occlusion of the internal carotid artery at its origin, while the external carotid artery filled well.

Frøvig [32] (1946) described a patient who had dimness of vision or "black-outs," especially when walking. Vision gradually deteriorated, and it is not clear from the report whether the defect was central or peripheral in origin.

Walsh [33] (1946), in his textbook, described a woman aged 43 who one day had spastic flexion of the fingers of the right hand. On the following day there were severe weakness of the right hand and less pronounced weakness of the right leg and foot. A sensation of spots in front of the left eye was complained of, as well as occasional diplopia. In the two weeks after the onset several episodes of total blindness occurred, lasting a few moments and affecting only the left eye. Pulsation was absent in the left arm and the left side of the neck. Six weeks later vision was 20/15 in each eye. This case was undoubtedly one of occlusion of the carotid artery.

Sie-Boen-Lian [34] (1948) described the case of a man aged 34 who had had attacks of blurred vision in the right eye and short attacks of weakness of the left arm, as well as paresthesia of the left arm and the left side of the head. The inferior macular arteries, and later the superior ones, were converted into fine white lines. The appearance of the macular arteries changed frequently in the subsequent months. On one occasion a sudden change of two arteries into white columns took place during examination. Within a few seconds the affected vessels became normal again. This case might well be one of disease of the internal carotid artery.

Di Luca [35] (1948) noted that while pressing on the carotid sinus of a patient aged 50 with simple glaucoma the retinal arteries showed rapid emptying from the center toward the periphery. As soon as pressure was released, the empty section of the artery refilled. During compression, the patient noticed difficult respiration, blurred vision, tremor, and paresthesia of the limbs. The author attributed the clinical picture to angiospasm.

Without doubt, several of the cases just outlined were instances of occlusion of the carotid artery. Although not numerous, they furnish strong corroborative evidence for the syndrome I am describing.

GROUP 2.—*Cases in the younger age group with no apparent cause.*

Homburger [36] (1864) described his own attacks of partial blindness, three in number, in which a cloud suddenly passed between him and the paper he was reading. In a few moments it expanded and enveloped him in a kind of semiobscurity, of a dark gray-greenish color. He could not see his hand before his eyes. Suddenly, after "some minutes," vision returned within a few seconds. Clearing took place from above downward.

Loring's [5] patient, at the age of 2 years, had a severe attack of rheumatism. At the age of 19 he had another attack, which invalided him for three years. At the age of 20 he began to have repeated attacks of transient blindness in the left eye. Blindness usually lasted a minute or two and recurred at variable intervals, sometimes daily, or there might be no attacks for weeks or months. The attacks were too numerous to keep count of. They had continued for about 24 years, when, at the age of 47, what seemed to be an ordinary attack came on, but blindness persisted. Examination showed that vision was reduced to light perception. The retinal arteries appeared normal, but the veins were distended. The retina was milky-white. Six weeks later the retinal vessels had become thread-like.

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32. Frøvig, A. G.: Bilateral Obliteration of the Common Carotid Artery: Thrombangiitis Obliterans? Acta psychiat. et neurol., Supp. 39, pp. 3-79, 1946.

33. Walsh, F. B.: Clinical Neuro-Ophthalmology, Baltimore, Williams & Wilkins Company, 1947.

34. Sie-Boen-Lian: Spasm of Macular Arteries, Arch. Ophth. **39**:267 (March) 1948.

35. di Luca, G.: A Provoked Retinal Angiospasm, abstracted, Am. J. Ophth. **31**:1190, 1948.

36. Homburger, J.: On Epilepsy of the Retina and Its Connection with Glaucoma, Am. J. Ophth. **2**:1, 1864.

Ramorino[37] (1877) described the case of a man aged 35 who for about 11 months had had attacks in which overlapping circles moved in from the temporal side and, on reaching the center, caused temporary blindness. At the onset the circles were not colored, but later they became all colors of the rainbow. The patient also had distortion of vision, in which the lines between pavement blocks enlarged and made it difficult to walk. When the attacks first began, there was some headache. Examined during an attack of blindness, the retinal arteries and veins were empty. When the blood returned, vision was restored. After the use of quinine the attacks stopped. The attacks were of several minutes' duration, and they apparently came at regular intervals. During the observed attack the face and ears became red. Ramorino's second case was that of a woman aged 40 who had had frequent attacks of blindness, which were cured by quinine. Headache was not present. The author attributed the phenomenon to "spasm of the tone of the retinal vessels."

Smith[7] described two cases. Case 4 concerned a woman aged 25 who for six months prior to examination had had a craving for food and who felt giddy and sick an hour or two after breakfast. At first vision was only dimmed during an attack, but in later attacks the sight had been lost entirely for half an hour. Six weeks before admission she had a particularly severe attack. Half an hour later sight came back to the left eye, but only to the lower half of the right eye. She had noticed that in the left eye sight always returned from above downward, but in the severe attack recovery in the right eye was in the opposite direction. Direct ophthalmoscopy revealed no change in either retina. Case 5 was that of a girl aged 18 who had had, in a period of three years, about six attacks of dimness of vision in the right eye, during which the lower half of objects was lost. The attacks came on suddenly, and as vision was lost the pupil dilated. Examination revealed nothing remarkable.

Doyne[38] (1889) described the case of a man aged 22 who while reading became totally blind in the right eye. In 10 minutes vision began to return from below upward, as well as he could remember, and in 5 minutes sight was completely restored. Several months later he experienced the same sensation in his right eye, but then it began to clear only after an hour and a half, and on this occasion from above downward, contrary to the way in which he believed the previous attack had passed off. The upper half of the retina showed a thick white fog two days later, and there was a corresponding complete loss of vision in the field below the horizontal. No plugging of the retinal arteries existed, but the finer branches were much more manifest than usual. Several months later the ascending arteries were much narrowed on the disk.

Noyes[39] (1890) described a woman aged 43 who, three months previously, began to have partial obscuration of sight in the right eye. The attacks occurred about once a day and lasted 15 to 30 minutes. Then, for three successive evenings, episodes of blindness occurred, lasting a few moments to half an hour. Observation of the left retina during an attack showed that the arteries were reduced in size, becoming larger as vision was restored.

Benson[40] (1894) described the interesting case of a man aged 32 who four years previously, for the first time, had had sudden complete obscuration of the left eye, lasting for a few minutes. Since then he had had similar attacks, sometimes affecting the whole field of vision, but most frequently implicating only one section of the field. At first they occurred only about once a month or so, but they gradually increased in frequency until he was having two a day. Between attacks vision had been good until two months before, when, over a period of a week or 10 days, vision decreased until he could not read at all. Examination showed vision to be 6/36 and 6/60 in the right and left eyes, with partial and complete central scotomas, respectively. The retinal veins pulsated strongly. On three occasions the affected eye could be examined during an attack. In one of these the largest division of the inferior temporal artery of the retina was entirely bloodless for about 4 disk diameters. The other vessels seemed normal. In a very few seconds the anemic portion of the vessel changed position,

37. Ramorino, D.: Due casi di iscemia dei vasi retinici, Ann. ottal. **6**:25, 1877.

38. Doyne, R. W.: Recurrent Transient Blindness, Tr. Ophth. Soc. U. Kingdom **9**:148, 1889.

39. Noyes, H. D.: Diseases of the Eye, New York, W. Wood & Co., 1890, p. 638.

40. Benson, A. H.: Recurrent Temporary Visual Obscurations, Edinburgh, Tr. Eighth International Ophthalmological Congress, 1894, p. 81.

moving toward the periphery of the fundus, at times slowly and steadily and at other times in less regular progressions, until it reached the next large bifurcation, when it suddenly disappeared completely, leaving the fundus in its usual normal condition. The patient stated that the dimness usually began at the outer side, then spread toward the center from all around until the eye was totally blind, the complete blindness lasting from two to five minutes. At times the blur began on the nasal side, either below or above. The right eye became affected in the weeks following his first examination. The dimness of the right eye began at the inferior temporal side and disappeared at the superior nasal side. There were never any associated complaints. Slight pressure on the globe of the eye produced marked pulsation in the central vessels of each eye. The patient kept a record of his attacks for about three months; he had 46 attacks—34 partial and 12 complete, 9 occurring on the right side and 37 on the left. A clinical assistant who examined the patient during an attack found the inferior temporal artery bloodless, corresponding to an upper field defect. Benson attributed the phenomenon to spasm.

Beevor and Gunn[41] (1899) described intermittent blindness in a brother and sister. The man, aged 34, had since he was 7 years old noticed attacks of blindness for a few minutes, the loss of sight affecting chiefly the right eye. During the attacks he saw brownish disks and zigzags without color, and they were not associated with headache. He believed that sometimes the left eye was attacked. Recently he had had an attack in which he suddenly saw zigzags and dark splotches and in a few seconds became blind in the right eye. The blindness which was so complete that he could only see a candle in the center of the field, lasted from afternoon to the next morning, when he observed that he had lost the upper half of the field of vision in the right eye. The condition persisted. In the following three months he had five attacks, and in each attack there was total blindness of the right eye. The appearance of the blindness, when it then occurred, was like an opaque thundercloud which suddenly came across the vision of the right eye and gradually broke up and dispersed, as a rule obstructing the sight for a minute or so. Subsequent attacks varied in frequency from two or three a week to two a month and affected only the right eye. Independent of these attacks, he had had, ever since he was at school, curious feelings of being tired and dreamy, lasting a few seconds. In the dreamy attacks he did not lose his senses but did not quite remember why he was in a certain place; occurring events did not seem natural, and he had the idea of impending calamity. These dreamy states occurred sometimes after the attacks of blindness. Occasionally he had had a sensation of needles in the left hand and foot for a few days. Examination showed complete loss of vision in the field of the right eye above the horizontal line and considerable diminution of peripheral vision below the horizontal line. On ophthalmoscopic examination of the right eye, the lower half of the optic disk appeared pale, and there was evident shrinking of the lower branches of the central retinal artery, the nasal horizontal branches showing thickened sheaths and the descending branches being of small caliber and appearing as white lines with considerable irregularity in one of them. The upper vessels and the lower veins appeared normal. The authors emphasized that for 24 hours after an attack a corresponding loss of field for green was found, although the fields were perfect for white.

After the failure of vision in the right eye, he had occasionally had specks and clouds before the left eye, and in one attack he lost sight completely for a few seconds in the lower half of the visual field of the left eye. Some time later, after being exposed to cold and fatigue, the patient had a curious attack, the nature of which was uncertain, in which he described the symptoms as coldness and numbness of the hands and feet, followed by tonic spasm of the limbs and then by general twitching, but without loss of consciousness.

The patient had a sister who for six years had had, three or four times in the year, curious attacks, which were described as follows: "She has a feeling of contraction or sometimes sharp pain at the back of the right eye and objects appear to recede from her and get smaller and finally to disappear in a white fog for a few minutes; the objects then rearrange

41. Beevor, C. E., and Gunn, R. M.: A Case of Obliteration of a Branch of the Retinal Artery, Following Frequent Attacks of Temporary Amblyopia, Tr. Ophth. Soc. U. Kingdom **19**:75, 1899.

themselves and in from ten to fifteen minutes from the commencement of the attack they return to their normal appearance." The attack was definitely unilateral and was not associated with headache, although she had been having attacks of true migraine about once a month, consisting of pain on the right side of the head and over the brow of the right eye. Those episodes were not associated with visual sensations, and the two types of attacks seemed to be distinct.

Cant [42] (1899) referred briefly to the case of a woman aged 22 who for years had been temporarily losing vision in the right eye. Attacks occurred about once a week and lasted 10 to 15 minutes. The eye gradually became blind, and a cherry-red spot appeared at the macula. The left eye became similarly, but temporarily, affected, and on one occasion a retinal hemorrhage was found.

Posey [12] described four cases which fall in this group. Case 2 was that of a man aged 30 who was seen because "he had noticed on two occasions that the lower half of objects became suddenly obscured. No headaches followed the loss of sight, though he thought he remembered seeing a play of lights before the eyes. He was unable to say whether both eyes were affected or not. A month later he had another attack in which the right eye became absolutely blind for fifteen minutes. There were no other motor or sensory symptoms and headache and nausea were absent. Three months after this, after reading he noticed a dark spot the size of a fifty-cent piece over the right eye when he regarded the flame of a lamp. The spot for a time was directly in the center of vision and then became eccentric. After persisting half an hour this phenomenon passed slowly away. One month later the upper half of his right eye became totally blind for fifteen minutes. Vision was then undisturbed for six months, when the lower half of the same eye became blind for five minutes. Nine months later the right eye became totally blind again, the loss of vision beginning in the lower part of the eye . . . . A week later he had another attack of dimness in the same eye, the loss of sight being more marked in the upper nasal field. During the last three years he had had several other attacks of blindness in the right eye the loss of vision usually beginning in the lower part of the eye." Posey's Case 3 was that of a man aged 50 who had seven attacks of monocular blindness in a period of 16 months, each attack lasting from 5 to 60 minutes. On four occasions the right eye was affected; on three, the left eye. The only abnormality noted on examination was unusual grayness of the optic disks. Case 5 was that of a man aged 20 who had attacks of blindness, affecting the right eye only, and coming once a month, for several years. Each attack lasted from 5 to 10 minutes. Posey's Case 7 was that of a man aged 39 who had had repeated attacks of blindness in the left eye since he was 8 years old, the attacks lasting from 5 to 10 minutes. Finally, after one attack blindness persisted.

Case 2 reported by Lundie [14] was that of a woman aged 22 who for eight years had had occasional attacks of loss of sight in one eye, which came on without traceable cause, were not associated with headaches, lasted not more than five minutes, and usually passed off completely. There had been perhaps 20 attacks altogether, and in all but one the right eye was the one affected. After one attack she found that a defect of sight remained. Corresponding to a sector-shaped defect in the lower inner quadrant, the retina was hazy and the arterial branch small. Several attacks occurred subsequently, but none of them did any further damage.

Kuffler [43] (1912) referred to the case of a student who suddenly became blind in one eye after several temporary attacks of blindness in the previous week. The patient also had fleeting attacks of erythema of the hands and feet, and his mother had the same complaint. On examination, the amaurotic eye presented the picture of embolism. Later, the other eye showed periodic loss of vision.

Hunter (1914), in a discussion on Langdon's [44] paper, described a confrère who suddenly said he could not see. Examination showed that the retinal blood stream was broken up "like the mercury column in a thermometer that had been dropped." Duane (1914), in a discussion on the same paper,[44] described the case of a woman who three years before, on two successive days, had had an attack of transient obscuration of the right eye. There was no

42. Cant, in discussion on Beevor and Gunn.[41]

43. Kuffler, in discussion on Weiss.[16]

44. Langdon, H. M.: A Case of Alternating Transient Monocular Blindness Ending in Complete Loss of Vision in the Left Eye, Tr. Am. Ophth. Soc. **13**:796, 1914.

abnormality in the retina. Two years later she had an attack in the other eye, and while central vision was still severely depressed the author found that a small cilioretinal artery running to the macula was totally occluded and thread-like. Upon the inhalation of amyl nitrite, the vessel was seen to dilate, and vision returned practically to normal.

Ormond [45] (1918) described the interesting case of a French army officer who for about 20 days had one or two daily attacks of transient blindness of the right eye, lasting about one minute and unassociated with headache or high blood pressure. During an attack, the pupil dilated, the optic disk became pale, and the retinal arteries disappeared, but the retinal veins in part remained visible. The attacks ceased after about three weeks.

Onfray [46] described the case of a young patient who repeatedly lost vision in the right eye, a field defect finally persisting.

Friedenwald [20] described the case of a married woman aged 21 who had had 10 or 11 attacks of blindness in the right eye, lasting a couple of minutes and described as being like a veil which came before the eye.

Bollack [47] (1926) referred to the case of a man aged 32 who for a dozen years had had monthly attacks of bilateral total blindness. After two minutes vision returned in the superior field of both eyes, and, after persisting thus for one minute, the visual field gradually enlarged downward. There were no associated symptoms, but on walking he was liable to dizziness and lateropulsion.

Haïri [48] (1926) described the case of a woman aged 23 who had had periodic attacks of blindness or simple scotoma in the right eye for eight years. Attacks occurred once every one to four weeks and lasted two to three minutes. After one attack, which was preceded by a severe headache, a superior nasal field defect persisted, corresponding to obstruction of the inferior temporal branch of the central retinal artery. The attacks continued thereafter, but less frequently.

The patient of Wagener and Gipner [21] (1927), a woman aged 20, after having had several attacks of blurred vision accompanied by a sensation of flooding of the eyes with light and numbness around the eyes, awakened to find she was blind in the left eye. Vision was 1/60; the optic disk was pale, and the retinal arteries were all small or completely obliterated. She had had no headaches.

Aubaret [49] (1929) described the case of a young actor aged 23 who had had three attacks of complete blindness within a few days. Blindness lasted 36 hours after the last attack, and recovery was attributed to inhalation of amyl nitrite.

The case of Cuénod, Nataf, and Bruguairolle [50] (1930) was that of a man of unstated age who for several years had been having sudden attacks of blindness in the left eye. They lasted up to six minutes and occurred once, twice, or several times a week. Surgical correction of a deviated nasal septum resulted in cure; at least, no attacks had occurred thereafter for six months.

Griffith [23] (1929) said that he had seen two physicians with spasm of the retinal artery. Both were convalescing from influenza. One became blind for two minutes in one eye. The other physician was approaching his car when it appeared that his wife, who was in the car, had no head. The attack soon passed off.

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45. Ormond, A. W.: A Note on the Direct Observation of the Fundus Oculi During a Period of Temporary Blindness, Brit. J. Ophth. **2**:273, 1918.

46. Onfray, R., in discussion on Bailliart, M.: Un cas de cécité intermittente par spasme vasculaire, Bull. Soc. opht. Paris **36**:154, 1924.

47. Bollack, M. J.: Accès de cécité transitoire suive d'hemianopsie horizontale, Bull. Soc. opht. Paris **38**:559, 1926.

48. Haïri, H.: Le spasme de l'artère centrale de la rétine et l'obstruction consécutive de l'artère temporale inférieure, Ann. ocul. **164**:662, 1926.

49. Aubaret, M.: Sur un cas de cécité par angiospasme rétinien, Rev. d'oto-neuro-opht. **5**:370, 1929.

50. Cuénod; Nataf, R., and Bruguairolle: Angiospasme rétiniens liés à une insuffisance nasale, Bull. Soc. opht. Paris **42**:373, 1930.

De Saint Martin[51] (1931) referred to the case of a man of 20 who during the previous year had had several attacks of sudden blindness in the left eye, lasting several minutes. The episode which brought him to the oculist was similar to the previous ones except that the blindness persisted, taking about 26 days to recover. The disk was pale, and the vessels were attenuated. Two months later a similar attack occurred, with considerable, but incomplete, recovery.

Guillot[52] (1933) described the case of a man aged 34 who, after having several temporary obscurations of vision in the left eye, suddenly became blind in that eye. Papilledema was present, and recovery was slow and incomplete. Vision had suddenly been lost in the right eye eight years before.

Traquair's[53] (1933) first case concerned a man aged 33 who began to have attacks of sudden temporary blindness or impaired vision at the age of 21. Altogether, he had had about 50 attacks in the right eye and only three in the left. The duration of an attack was five to seven minutes. The attacks were liable to occur when he was bending down and were more frequent in wet weather. There were no other symptoms.

Kravitz[54] (1934) described two cases. The first was that of a man aged 31 who had had numerous attacks of blurred vision, lasting a minute or two. In one attack vision in the left eye remained blurred, and everything in the left temporal field was indistinct. The superior nasal artery of the left eye was contracted, and a slight retinal haze surrounded it. Inhalation of a vasodilator resulted in almost immediate return to normal. His Case 3 was that of a man aged 43 who for three years had had attacks of blindness in the right eye, lasting a few minutes. One attack did not clear so promptly, and one day later blindness persisted and the right inferior temporal artery was found to be very thin. One month later the patient had an attack in which the upper half of all objects was invisible. Aubaret and Jayle[55] (1934) referred to a girl of 17 who was having episodes of total blindness, lasting 10 to 15 minutes and recurring about once a week.

Sédan and Gaëtan[28] described the case of a young girl subject to sudden blindness in the right eye, lasting from a few seconds to a minute and occurring several times a month. The authors attributed the attacks to latent epilepsy. They described another case in which attacks of blindness took place in the interval between epileptic seizures.

Brunner[56] described the case of a man aged 34 who was subject to sudden temporary blindness in the left eye. Observation during an attack showed that the retina and optic disk were pale, the arteries narrowed, and the circulation arrested. The blood column in the vessels was fragmented.

Lacat[56] briefly referred to the case of a physician who suddenly lost the vision of the right eye for several minutes.

These cases have many common features, and yet it is difficult to classify them further, for the frequency, duration, and sequelae are all highly variable. Blindness limited to one portion of the visual field of one eye appears to be not uncommon, and examination usually shows attenuation of the corresponding vessels. Visual defects are as a rule in a horizontal, rather than a vertical, plane. Evidence of vasomotor instability in other parts of the body is, as a rule, lacking.

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51. de Saint Martin: Un nouveau cas d'angiospasme rétinien diagnostique et guéri par l'acetylcholine, Bull. Soc. opht. Paris **43**:28, 1931.

52. Guillot, M. P.: Angiospasme de l'artère central de la rétine à forme d'oedème de la papille, Rev. d'oto-neuro-opht. **11**:193, 1933.

53. Traquair, H. M.: Spasm of the Retinal Artery, Tr. Ophth. Soc. U. Kingdom **53**:118, 1933.

54. Kravitz, D.: The Vasomotor System in Retinal and Cerebral Vascular Lesions, Am. J. Ophth. **17**:741, 1934.

55. Aubaret, E., and Jayle, G. E.: Crises brusques et transitoires de cécité complète bilatérale d'origine périphérique chez une jeune fille, Rev. d'oto-neuro-opht. **13**:217, 1935.

56. Cited by Sédan and Gaëtan.[28]

GROUP 3.—*Cases associated with migraine.*

Testelin[57] (1866) described the case of a girl aged 10 years who had had repeated attacks of blindness in the left eye occurring twice a day, at 11 a. m. and about 4 p. m. The attacks lasted 15 to 30 minutes and were heralded by slight malaise, "a sort of chill," a little headache, and increased warmth of the left cheek. A diagnosis of malaria was made; quinine was prescribed, and the attacks ceased. This interesting case is placed in the migraine group, as the clinical picture does not seem to justify the diagnosis of malaria and each attack was associated with headache.

Hutchison[58] (1871), in his paper on xanthelasma palpebrarum, included the case of a man aged 45 who for years had been subject to sick headaches, often preceded by temporary blindness in one or the other eye, lasting a few minutes. In the last attack vision in the right eye was lost permanently.

Konigstein[59] (1875) carefully studied a woman aged 31 who lost vision periodically in the right eye. Attacks occurred in the evening, lasting a few minutes, and were associated with headaches and supraorbital "spasms" on the right side. Quinine was prescribed, and seemed to abolish the attacks.

Landesberg[60] (1878) described the case of a man aged 25 who had had several attacks of blindness of the left eye, each attack lasting "hours" and followed by left-sided headache.

Nettleship[6] (1879) reported the case of a woman aged 26 who, after having transient attacks of dim vision in the right eye, awakened one morning with blindness on that side. Thereafter, similar fluctuations in vision began on the other side. She had had rheumatic endocarditis and was also subject to headache. This case might well be classified in the cardiac group.

Galezowski[61] (1882) described three cases of blindness associated with headache. Case 1 was that of a general aged 67 who had had typical migraine associated with scotoma for more than 20 years. He suddenly lost vision in the right eye, examination revealing attenuated vessels characteristic of thrombosis of the central retinal artery. Case 2 was that of a girl aged 15 who had had migraine since the age of 7 or 8 years. She would suddenly have a headache, preceded by a scotoma or zigzags in one or both eyes. Sometimes vision was lost for 10 or 15 minutes. The attacks occurred at variable intervals but for one year had occurred every day. One day the aura gradually passed into total blindness of the left eye. In the preceding months the scotomas had almost always occurred in that eye. The retinal vessels were attenuated, and the diagnosis of thrombosis of the central retinal artery was made. Case 3 was that of a woman of 29 who, after confinement four years previously, began to have severe headaches, preceded by watering, burning, and bright zigzags of different colors in both eyes. For four years the zigzags and dancing scotomas had been constant. Examination showed optic nerve atrophy on the left side.

Smith[7] reported two cases in this group. The first was that of a woman aged 40 who for three or four years had had repeated attacks of total blindness in one or the other eye, lasting sometimes for half an hour, but oftener for a few minutes only. She always had a severe headache after an attack. Three days before admission she felt queer in the head and then suddenly became blind in the right eye. She sat down and waited for sight to return, but blindness persisted. On examination, the retina presented the appearance considered typical of embolism, the arteries being extremely small and empty. General examination showed cardiac enlargement and aortic obstruction. Again, heart disease may have been a factor in this case. The second case concerned a woman aged 20 who suddenly lost the sight in the right eye for everything below the level of the point looked at. She said she had had occasional attacks of loss of sight, lasting 5 or 10 minutes and followed by headache. She thought only one eye was affected in the

57. Testelin: Fièvre larvée double quotidienne; forme amaurotique, Ann. occul. **56**:317, 1866.

58. Hutchison, J.: A Clinical Report on Xanthelasma Palpebrarum, and on Its Significance as a Symptom, Med.-Chir. Tr. **54**:171, 1871.

59. Konigstein, L.: Ein Fall von täglich wiederkehrender totaler einseitigen Amaurose, Klin. Monatsbl. Augenh. **13**:333, 1875.

60. Landesberg, M.: Zur Pathologie des Sympathicus, Arch. Augenh. **7**:268, 1878.

61. Galezowski, X.: Migraine ophthalmique, Rec. d'opht. (Series 3) **4**:10, 1882.

attacks, but did not know which. The upper division of the retinal artery was reduced to fine threads, one branch being quite white, the other showing a very thin red line. There was a loud mitral murmur in this case.

Morton [62] (1890) described the case of a woman aged 22 who for two years had had periodic attacks of blindness in the right eye. The blindness, which was preceded by pain in the right temple and brow for three or four hours, would come on within three minutes and last about 10 minutes as a rule. Occasionally the upper half only of the field was obscured. During the attack she felt cold and sometimes giddy, but not faint. The day before being examined, she had had an attack in which the upper half of the field in the right eye became dim, and within five minutes vision was completely lost in that eye. Sight did not return, as on former occasions, and ophthalmoscopic examination showed all the appearance of obstructed retinal circulation, although even after 30 hours the arteries could be made to pulsate by firm pressure. Later, similar attacks occurred in the left eye, in one of which vision failed from above downward and returned from below upward. The patient had had chorea four years previously, but there was no evidence of heart disease.

Berger [63] (1897) referred to a woman aged 36 who for 20 years had had left-sided headaches. Finally, attacks of anesthesia and paresis occurred on the left side, and total blindness of the left eye appeared during the night. The nerve head was pale, and the arteries were attenuated. Paracentesis produced a remarkable return of vision.

Thomson [64] (1902) had as a patient a woman aged 34 who for 10 years had been subject to periodic paroxysms of sick headache, vomiting, and, usually, misty vision in the right eye, the last symptom lasting from 5 to 10 minutes. After one attack a black mist persisted before the right eye. Examination showed narrowing and poor filling of the superior retinal arteries, as well as a damaged retina. Vision partially returned after 2½ months.

Langdon [44] (1914) had a patient, aged 40, who complained of severe generalized headaches, occurring once a week and lasting 24 hours. Monocular blindness also occurred, the attacks lasting three seconds to five or six minutes, the eyes being about equally affected. Only once had the patient been blind in both eyes at the same time, the attack lasting about two minutes. He also had had transient diplopia, occurring about once a day, one object being above and to the right of the other. The episodes usually lasted about 30 seconds. Often the first thing he would notice in an attack of blindness was diplopia; vision would then gradually become hazy until it was entirely gone; then a hole would appear in the fog, as he expressed it, and vision would gradually return. Central vision always seemed to return before the peripheral field cleared. He had never had attacks of hemianopsia or scotoma scintillans, nor did a headache accompany the blind spells. The blood pressure was 140 mm. Nine years later, he returned because an attack had begun the previous evening at 8 o'clock and had not cleared, the eye still being blind. The fundus on the affected side showed that the retinal arteries were reduced to threads. The patient later had heart disease, characterized by an aortic regurgitant murmur, great cardiac dilatation, and peripheral edema, from which he recovered completely. The left eye remained blind. This case is included in this group, although it cannot be considered an ordinary case of migraine.

Bruner [65] (1921) described the case of a man aged 34 who for seven days had had repeated attacks (up to 10 a day) of blindness in the left eye, lasting a few minutes. He had had slight frontal headaches, and at times he would awaken with a dull pain in his head, sometimes accompanied with dazzling sensations in the eyes. During the attack of blindness, the disk and retina of the left eye became pale; the arteries were reduced to threads, and the veins showed complete cessation of blood flow. The blood in the veins assumed a granular appearance, owing to the breaking up of the normal blood column. In from one to three minutes the flow of blood in the veins recommenced, at first slowly and gradually returning to normal. Attacks ceased after the instillation of physostigmine and reduction in the intraocular pressure.

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62. Morton, A. S.: Obstructed Retinal Circulation, Ophth. Rev. **9**:65, 1890.

63. Cited by Wagenmann.[8]

64. Thomson, G. W.: A Case of Obliteration of a Retinal Artery Following Attacks of Temporary Amblyopia, Tr. Ophth. Soc. U. Kingdom **22**:177, 1902.

65. Bruner, A. B.: Spasm of the Central Retinal Artery, Am. J. Ophth. **4**:503, 1921.

Löhlein [66] (1922) described the case of a man aged 42 who had severe migraine associated with transient amaurosis, for 20 years. Hemianopsia had never been noted. Vision was lost permanently in the left eye during an attack. Later the right eye was similarly affected, but its vision recovered to 3/8. The retina showed hemorrhages.

Bailliart [67] (1924) reported the case of a woman aged 34 who had had severe migraine since infancy. In 1921 she had had one attack; in 1922, three attacks; in 1923, three attacks, and in 1924 one attack, all followed by sudden bilateral blindness. Each attack lasted three to seven days, but later it was found that inhalation of amyl nitrite would alleviate such an attack almost immediately. Blindness in this case was probably central in origin. Friedenwald [20] described the case of a woman aged 34 who had had attacks of flickering, characteristic of migraine, for 10 years. However, in 1921 and 1922 she had four or five attacks in which only the right eye became blind—except for light perception—for 10 to 15 minutes. She had five attacks in 1923, one in 1924, and none for two years thereafter. It is not clear that the patient had typical migraine at any time.

Lister [68] (1931) referred to a case in which obscuration of vision took place suddenly in one eye. Immediate examination showed blocking (spasm) in both the superior and the inferior retinal artery. Later the vessels were normal. The patient was subject to migraine.

Law [23] reported the case of a girl who had had three attacks of obscuration of the upper half of the field in one eye, accompanied with headache. The inferior temporal artery was empty, and there was a corresponding scotoma.

Kurz [69] (1932) referred to a man aged 34 who had had migraine for several years. For two months he had had attacks of greatly blurred vision, lasting a few minutes. On examination during an attack, blood was seen only in the arteries near the equator, but suddenly the arteries filled with blood.

Traquair [53] described the spectacular case of a man aged 55 who had had attacks of unilateral headache, preceded by seeing zigzag lights, since the age of 13. The headaches were sometimes on one side and sometimes on the other. At about the age of 32, he began to see, occasionally, transparent obscurations and distortions, described as a filmy landscape, before his eyes. These attacks occurred several times yearly, and after each he perspired and his face went "ashy-gray." At about the age of 38 "clouds" began to form, sometimes in front of one eye and sometimes in front of the other. About the same time, he began to have attacks of whiteness of the index fingers, which became cold and numb for about 15 minutes. When he was 52, one of these clouds before the right eye became permanent. Between the ages of 51 and 54, he had 34 attacks in the right eye and 43 in the left eye. The average duration was four minutes, but attacks lasted 10, 13, 32, and 55 minutes, respectively. Results of examination were not remarkable except for a small scotoma skirting the fixation point. This case, too, is far from a straight-forward instance of migraine.

Quick [70] (1932) stated that he himself, 30 years before, had suddenly become blind in the left eye for a few minutes. He had been subject to migraine, but this blindness was different from the migraine scotoma and much more terrifying. At times his fingers became numb, sometimes as part of the migraine, at other times accompanied by pallor of the parts.

Grönvall [71] (1938) gave a detailed description of the case of a girl aged 18 who for four or five years had been having migraine headaches, preceded by dimness of vision and large grayish-white patches with a colored margin before her eyes. Finally, she suddenly became blind in the right eye, but within 10 minutes she began to see objects in the lower part of the visual field.

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66. Löhlein: Erblindung durch Migräne, Klin. Monatsbl. Augenh. **68**:390, 1922.

67. Bailliart, M.: Un cas de cécité intermittente par spasme vasculaire, Bull. Soc. opht. Paris **36**:154, 1924.

68. Lister, W., in discussion, Tr. Ophth. Soc. U. Kingdom **51**:121, 1931.

69. Kurz, J.: Spasms of the Retinal Vessels, Oítal. Sbornik **6**:188, 1931; abstracted, Am. J. Ophth. **15**:1003, 1932.

70. Quick, H. E., in discussion, Tr. Ophth. Soc. U. Kingdom **53**:118, 1933.

71. Grönvall, H.: On Changes in the Fundus Oculi and Persisting Injuries to the Eye in Migraine, Acta ophth. **16**:602, 1938.

When examined the following day, she still had a defect in the upper nasal quadrant and the adjoining part of the upper temporal quadrant of the right visual field. One branch of the inferior retinal artery was as "thin as a thread," and edema of the lower part of the retina was present.

Thus, there are but a few unequivocal cases of unilateral blindness associated with migraine. Evidence that the retinal vessels take part frequently, but less strikingly, in the migrainous process has long been sought, but the majority of observers have found no definite change in the caliber of the retinal arteries, even during visual symptoms. A few authors have reported vasoconstriction; others, dilatation. In some of the cases just mentioned in which the headache was of brief duration, tumor, aneurysm, arteritis, or syphilis might well have been responsible.

GROUP 4.—*Cases associated with Raynaud's syndrome.*

Raynaud [72] (1874), in his second paper, presented the case of a printer aged 59, who, in addition to periodic attacks of lividity of the fingers and hands (chiefly the left), had transient disturbances of vision. It was most unexpected to find that sight was good in both eyes during the period of abnormality of the fingers, but as color in the fingers returned to normal, the sight of the left eye became troubled and confused, recovering at the moment that a new peripheral attack supervened. While vision was affected and the hands were of normal color, ophthalmoscopic examination showed constriction of the retinal arteries adjacent to the disk, partial strangulation here and there, and, finally, a remarkable venous pulsation, all signs being more pronounced on the left side. Examination during the period of cyanosis of the fingers was less decisive; the arteries did not recover their normal caliber throughout their extent but presented partial diminutions of caliber, which in places rendered them filiform. By patient watching, contractions could be seen to appear and disappear. Cessation of the attacks of discoloration of the hand led to the cessation of visual complaint. Raynaud referred to this case when reinforcing his theory of vasospasm. His second case was that of a man of 22 in whom obscuration of sight had occurred at the same time as the attacks of cyanosis of the hands. During an attack, the retinal arteries were narrowed, becoming widened again as the attack passed off. Raynaud did not emphasize this case because it was complicated by the presence of a cardiac murmur and polyuria. Panas [73] (1894) later claimed to have made examination in Raynaud's original cases during and between attacks, but could find no difference in the caliber of the vessels. He also said that in one case blindness improved during a cold bath but reappeared soon after.

After Raynaud's paper, scattered reports of ocular changes in Raynaud's disease appeared. Dunphy [74] (1932) reviewed the entire literature and concluded that it was doubtful whether it had ever been shown that Raynaud's disease had involved the retina, the cases always being complicated and highly atypical. He quoted Osler as saying, "I have looked in vain for signs of constriction of the retinal vessels in several typical cases." Dunphy then described a most unusual case of a girl aged 9 years with an illness characterized by arthritis, a cutaneous rash, and fever. Over a period of five months improvement occurred, but she then complained of poor vision in the right eye. The retina showed a definite red papule lateral to the disk, with the rest of the retina pale. Six months later cyanosis and coldness of the finger tips and toes had developed. The tips of the third and fifth finger on the right hand became black and gangrenous, and the second and third fingers of the left hand were cold and painful. At about this time she suddenly lost vision in the left eye. On examination half an hour later the whole retina was blanched, with a cherry-red spot standing out in the macular region. The arteries were extremely thin and contained broken columns of blood moving slowly away from the disk. In a short time this movement became exceedingly slow; in fact, at times the red masses seemed to move first forward and then backward, without making any definite progress. The disk

72. Raynaud, M.: New Researches on the Nature and Treatment of Local Asphyxia of the Extremities, Arch. gén. de méd. **1**:5, 189, 1874; translated by T. Barlow and published in Selected Monographs, London, New Sydenham Society, 1888.

73. Panas, P.: Traité des maladies des yeux, Paris, 1894, Vol. 1, p. 619.

74. Dunphy, E. B.: Ocular Manifestations of Raynaud's Disease, Tr. Am. Ophth. Soc. **30**: 420, 1932.

margin became blurred, owing to edema of the surrounding retina. Inhalation of amyl nitrite, corneal paracentesis, use of glyceryl trinitrate, and cervical sympathectomy did not give relief.

Previous reports of intermittent blindness associated with Raynaud's disease included one by Fisher [75] (1902). He briefly referred to a patient with recurrent symmetrical syncope of the fingers in cold weather, who also had frequent attacks of temporary obscuration of vision. Bailliart [67] described a case in which there had been several attacks of bilateral blindness over a period of three years. One attack, while the patient was under observation, was preceded by headache for eight days, orbital pain, and chilliness. The latest attack had been accompanied with pallor of the face. The disks were pale and the vessels narrow. Inhalation of amyl nitrite restored vision immediately. The hands showed changes resembling Raynaud's syndrome.

Appelbaum and Lerner [76] (1926) described an unusual case of a woman aged 45 who first had attacks of pallor and congestion of the fingers of the left hand and later, attacks of "migraine," associated with temporary loss of memory, numbness, pricking of the fingers, feet, and tongue, and disturbed vision. Later, bilateral papilledema appeared and persisted for several months.

Wagener and Gipner [21] described the case of a woman aged 45 who had a moderate degree of arterial hypertension. For 10 years she had been having short periods of blindness, more frequently in the left eye, sometimes complete, at other times involving only a part of the visual field. At times the loss of vision occurred in one eye; at others it was in both eyes, but never complete in both eyes at the same time. In addition, her fingers became white on exposure to cold, and she had peculiar spells, lasting two or three minutes, of mental confusion without loss of consciousness. The first such attack came on after a cold swim. For two years she had had nocturnal seizures. While being examined, she suddenly noticed loss of vision in the superior nasal field of the left eye, and it could be seen that the main secondary branch of the inferior temporal artery terminated abruptly a short distance from its bifurcation. No trace of the vessel was visible beyond this point. As the authors had described in some of their other cases, "in three peripherally progressing pulses" the vessel became normal, and vision was restored. This case might well be placed in the group of atherosclerotic patients with hypertension.

Allen and Brown [77] (1932), in their review of 147 cases of Raynaud's disease, found 1 in which attacks of blindness suggestive of intermittent spasm of the retinal arteries had occurred. Anderson and Gray [78] (1937), and later Carpenter and Carpenter [79] (1938), reported the case of a man aged 39 who had had Raynaud's disease for 11 years. Gangrene of some of the toes and fingers on the right side had required amputation. The patient suddenly became blind in the right eye 13 days before examination, which disclosed the retinal arteries to be thin and small, several branches being bloodless. Broken columns of blood in the arteries could be seen moving slowly away from the disk. Examination by Carpenter and Carpenter, carried out some weeks later, showed that the right eye could discern only light, the pupil not reacting to light. The retina was pale; all the arteries appeared as white streaks; three secondary veins contained small segments of blood, and there was a red spot in the center of the macula. Suddenly, while the retina was under observation, a primary artery shot full of blood to the point where it divided. Then another arterial trunk and another showed this phenomenon, in lightning-like rapidity, the appearance suggesting the spokes of a cart wheel. The next change in the picture was a similar filling of the secondary arteries, and then of the veins. This phenomenon was repeated every few moments for over two hours. Over a period of weeks the frequency of repetition decreased. Vision was not recovered.

From this summary, it is apparent that ocular complications of Raynaud's syndrome are rare. Intermittent blindness is rarer still, and when it has occurred Raynaud's phenomenon may well have been a coincidence.

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75. Fisher, J. H., in discussion, Tr. Ophth. Soc. U. Kingdom **22**:177, 1902.

76. Appelbaum, S. J., and Lerner, M. L.: Raynaud's Disease with Ocular Complications, Am. J. Ophth. **9**:569, 1926.

77. Allen, E. V., and Brown, G. E.: Raynaud's Disease, J. A. M. A. **99**:1472 (Oct. 29) 1932.

78. Anderson, R. G., and Gray, E. B.: Spasm of the Central Retinal Artery in Raynaud's Disease, Arch. Ophth. **17**:662 (April) 1937.

79. Carpenter, W. M., and Carpenter, E. W.: Raynaud's Disease with Intermittent Spasm of the Retinal Artery and Veins: Follow-Up Report of Case, Arch. Ophth. **19**:111 (Jan.) 1938.

GROUP 5.—*Cases associated with heart disease.*

Knapp [10] described the case of a man aged 35 with heart disease, who had become blind in the left eye six years before. For two months he had been having transient attacks of dim vision in the right eye, lasting one-half to three hours.

Nettleship [6] referred to a patient aged 28 who had previously lost vision in the left eye, the retinal appearance suggesting that the attack had been a hemorrhagic episode. After that incident the patient began to have attacks of dimness of vision in the remaining eye. These attacks lasted from a few minutes to several hours and were accompanied with pain and headache. The patient had a very marked aortic obstruction and regurgitation. The author favored the explanation of a failure of blood flow, manifesting itself in the retina only. Barré and Duverger [80] (1923) described a case in which there was aortic stenosis and peripheral disturbances of the circulation. Two attacks of sudden transient blindness occurred, in which the vessels of the left eye became thread-like. The visual field remained slightly contracted, but there were no persistent visual symptoms. The attacks were attributed to spasm. Onfray [46] described a patient with mitral stenosis who had had five or six attacks of transient blindness in two years.

Rosenstein [81] (1925) referred to an unusual case, that of a man aged 28 with mitral stenosis who for eight years had had attacks of bilateral blurred vision, sometimes three or four times a day and lasting up to 45 minutes. Associated with the attacks were dizziness and neck ache.

Wagener and Gipner [21] described the case of a woman aged 23 with disease of the aortic and mitral valves. During examination a branch of the left inferior nasal artery was seen to terminate abruptly about 2 disk diameters from the disk. While this area was being studied, the vessel extended suddenly, as though with a pulse beat, for about ¼ disk diameter. A second pulse produced an approximately equal lengthening, and a third suddenly opened the vessel out to the periphery. The patient was not questioned about transient attacks of blindness. A Raynaud-like phenomenon of the right hand developed later.

The association of intermittent blindness and heart disease appears to be rare, but it is to be recalled that in a few cases placed in the migraine group heart disease was also present. Amaurosis is certainly not mentioned by cardiologists as an accompaniment of severe valvular disease. I have recently examined a man aged 42 with severe mitral disease who one year ago had about six attacks of blindness in the right eye. Each attack lasted 30 to 60 minutes and began with the sensation that a cloud with a sharp front advanced across the eye horizontally, from the medial to the lateral aspect. He has had no attacks for several months, and vision is perfect.

GROUP 6.—*Case of reflex origin.*

Smith [7] reported the case of a woman aged 24 who two years before had had a miscarriage and since then had suffered much from pelvic pain. She had been using a vaginal douche twice daily, and two months previously while using the syringe, she had caused herself intense pain and fainted. On recovering consciousness, she found that the left eye was quite blind. Since that occasion she had lost vision temporarily in the right eye three times, each time when the syringe touched an especially tender part, which always made her feel faint and ill. Blindness in the right eye lasted one-half to one hour, and sight seemed to her to return from the side. On examination, the right optic disk appeared pale and the retinal arteries shrunken to threads. Cardiac murmurs extended along the vessels of the neck, and these were attributed to the state of the blood. There followed in the next three months several short attacks of blindness, apparently brought on by exertion and fatigue. One morning she hurried to catch a train; on entering the carriage she fainted and did not recover her sight for some time. Another attack

80. Barré and Duverger: Troubles visuels à type embolie de l'artère centrale de la rétine, Presse méd. **31**:492, 1923.

81. Rosenstein, A. M.: Beitrag zu dem beiderseitigen Verdunkelungen des Sehvermögens mit vorübergehenden ophthalmoskopischen Befund bei Herzklappenfehler, Klin. Monatsbl. Augenh. **75**:357, 1925.

followed a painful defecation and lasted two hours. An abdominal section, with removal of both ovaries and both Fallopian tubes, abolished the attacks except for one related to painful defecation immediately after operation. Smith thought that loss of sight depended on reflex influences arising in the pelvis. He held it likely that failure of the circulation was the immediate factor; at the same time, localized spasm of blood vessels was a distinct possibility.

Parker [82] referred to the case of a middle-aged woman who partially lost vision of the right eye during amputation of the turbinate body on the right side, with nitrous-oxide anesthesia. The right pupil was dilated, and the upper half of the fundus had lost its color. The arteries were invisible, and there was blindness in the lower portion of the visual field. Two hours later she had recovered completely.

Wagener and Gipner [21] referred briefly to the case of a young woman of 20 who, while eating ice cream, had onset of blindness in the right upper visual field, with which there was associated obliteration of most of the corresponding inferior temporal artery. This case is included although the blindness was not intermittent.

Swett [83] (1935) reported an unusual case in which sharp pain and blindness occurred in the left eye while the right antrum was being irrigated. Examination three minutes later showed the fundus to be pale and the arteries completely empty. After a short period of massage, the arteries seemed to take on a rapid clonic dilatation and contraction, a column of blood advancing and receding with marked pulsation. As this phenomenon increased, it seemed to be transmitted to the vein, so that the whole disk pulsated for 20 minutes; then the pulsation grew progressively weaker, until the retinal arteries had completely refilled. The following morning vision was 20/20.

Hirsch [84] reported that while he was washing out an antrum the patient suddenly became blind in the eye on the side of the affected sinus. The pupil was dilated and the retinal vessels were emptied. Recovery occurred in two or three minutes. The author attributed the incident to air embolism.

GROUP 7.—*Miscellaneous cases.*

Moos [85] (1867) described a complicated case in which a loud heart murmur, jaundice, petechiae, and intestinal hemorrhage were present. The patient had three transient attacks of blindness, the first in the right eye, lasting one hour, and the next two apparently involving both eyes, everything appearing darkened. Transient diplopia had also occurred. Results of examination of the eyes are not reported. Smith [7] described the case of a man aged 38 who was awakened from an afternoon sleep feeling giddy and queer and found that he was blind in both eyes, except that he could see something like stars before his eyes. In a few minutes sight returned to the right eye, but the left eye remained nearly blind. Examination three years later showed a white disk and arteries so small as to be scarcely visible. Knies [86] (1888) referred to an epileptic patient aged 35 who began to have one-minute attacks of blindness in the left eye. The author wondered whether this could not have been the only evidence of a seizure resulting from vasoconstriction. Noyes [39] referred to a man who had had complete blindness in both eyes for 16 hours. The optic disks were pale and the arteries tenuous. Vision was restored 20 minutes after inhalation of amyl nitrite.

Mauthner [63] found severe anemia of the retinal vessels in a case in which fluctuating blindness had been present for 1½ hours. Vision returned while the author was seeking the necessary instruments to do an iridectomy.

Posey [12] referred to a young woman with epilepsy who claimed to have transient attacks of monocular blindness which was sudden and complete. Both eyes had been affected, but never at the same time. Another of his patients, a man aged 40, was seen because of sudden loss of sight in the right eye two days previously. The patient stated that 10 years before, while sitting at

82. Parker, W. R., in discussion, Tr. Am. Ophth. Soc. **13**:817, 1914.

83. Swett, W. F.: Reflex Spasm, Simulating Embolus of Central Retinal Artery: Secondary Glaucoma Following Antrum Irrigation; Complete Recovery, Am. J. Ophth. **18**:359, 1935.

84. Hirsch, C.: Luftembolie in die Arteria centrales retinae nach Kieferhöhlenspülung, Klin. Monatsbl. Augenh. **65**:625, 1920.

85. Moos: Beitrag zur Casuistik der embolischen Gefässkrankheiten, Arch. path. Anat. **41**: 58, 1867.

86. Knies: Augenbefund bei Epilepsie, Arch. Psychiat. **20**:569, 1888.

the table eating, he had suddenly become totally blind in both eyes. The condition persisted for two or three minutes, when vision slowly returned. Then for several years he had attacks of vertical diplopia, lasting for five minutes or so. Eighteen months before, he began to have spells of blindness in the right eye, coming on suddenly and lasting a few minutes. At times blindness was complete; at others, only partial. He had 15 or 20 attacks in all. He also had attacks of a curious smothering sensation when he talked. The present attacks began to clear in six or eight hours; when he was first seen, the disk margins were obscured, the retinal arteries, chiefly the upper ones, being attenuated.

Asmus [87] (1905) presented two cases of unilateral blindness with onset after blowing the nose. In one case vision returned to 6/6 in three days. Valude and Gallois [88] (1922) described the case of a woman aged 26 who suddenly became blind in both eyes. The right eye recovered in 15 minutes, but the left did not recover for about a month, and then only partially. Two further attacks occurred, but vision finally became normal. The patient was susceptible to cold, a bath producing asphyxia of the left lower extremity and the right arm. She often had cramps in the limbs. The Wassermann reaction of the blood was negative, and the blood pressure was normal.

Rosenstein [89] (1927) described the case of a woman aged 31 who for 14 years had had transient attacks of obscuration of vision at the time of her menstrual periods. The attacks were in either or both eyes, usually lasted only a few seconds, occurred 5 to 15 times a day, and were associated with headache, weakness, palpitation, and vomiting. The author examined the eyes on numerous occasions and found the pupil fixed and dilated, the retina pale, and the vessels constricted and empty. After each attack the patient was somnolent, so that it was suspected that the vascular spasm within the eye was part of a widespread change. One attack of blindness in the right eye lasted nine days, and only partial recovery took place, vision remaining 6/60. Endocrine therapy resulted in improvement. Rifat [90] (1929) described two cases of sudden loss of vision with attenuated vessels, in which atropine helped recovery. In one case transient blindness lasted three hours. Sédan [91] (1929) referred to a man who during the fever phase of malaria became totally blind. The retinal vessels were reduced to threads, and recovery took place over a period of seven days. The author regarded the case as one of vasospasm.

Nègre [92] (1929) referred to a man aged 73 who was subject to attacks of complete blindness, preceded by vertigo and precordial distress. The author witnessed one attack in which the patient became cadaveric and haggard, the skin became cold, and the pupils dilated as blindness appeared. Examination showed in all the arteries a dilated segment, which moved slowly along the vessel, leaving behind an exsanguinated, filiform white vessel. The blood pressure rose from 125/80 to 260/150. After several minutes the patient could see light again. In addition, he had had attacks of "dead finger," vague visual complaints, and bouts of furor. The attacks of blindness ceased after four years. Paroxysmal hypertension must surely have been responsible for the clinical picture in this case.

Davenport [93] (1931) described the spectacular case of a man aged 57 who, while being treated for bouts of stabbing, cramping pain in the limbs and neck associated with fever and difficulty in micturition, began to have sudden attacks of blindness in his left eye. These attacks lasted from a few seconds to three minutes, and as many as 14 occurred in one day. Later,

87. Asmus: Ein Fall von transitorischer einseitiger Amaurose infolge von Nasenschnauben, Deutsche med. Wchnschr. **31**:405, 1905.

88. Valude and Gallois, J.: Un cas de spasme artériel rétinien à recidives, Bull. Soc. opht. Paris **34**:20, 1922.

89. Rosenstein, A. M.: Weiterer Beitrag zu den beiderseitigen Verdunkelungen des Sehvermögens mit vorübergehenden ophthalmoskopischen Befund bei Herzklappenfehler während der Menses, Klin. Monatsbl. Augenh. **78**:248, 1927.

90. Rifat, A.: Deux cas de spasme de l'artère centrale de la rétine, Ann. ocul. **166**:711, 1929.

91. Sédan, J.: Cécité temporaire par angiospasme rétinien d'origine paludique, Ann. ocul. **166**:705, 1929.

92. Nègre, M.: Spasme total bilatèral des artères rétiniennes s'établissant sous l'ophthalmoscope, Bull. Soc. opht. Paris **41**:740, 1929.

93. Davenport, R. C.: A Case of Spasm of the Central Retinal Artery, Tr. Ophth. Soc. U. Kingdom **51**:115, 1931.

similar attacks began in the right eye. The author examined the eye during three episodes of blindness and found a fixed dilated pupil, a very pale optic disk, and the arteries so narrowed as to be hard to trace. The blood column in the veins was broken up "in the so-called cattle-truck way," the segments of blood moving jerkily in different directions in various parts of the system. As the attack passed, the arteries filled in a moment, and the blood column rapidly filled the veins. The patient became convinced that looking down was an unfailing way to start symptoms, and one of the attacks seen was voluntarily induced in that way. His general health gradually improved, and attacks ceased after five months. The author attributed the picture to arterial spasm. This case may well have been one of central retinal arteritis associated with polyarteritis.

Kurz[69] described the case of a nurse aged 29 who complained of blurring of vision in the right eye. There was apical tuberculosis, and a solitary tubercle of the retina was found. Repeated attacks of defect in the upper visual field occurred, after one of which the defect persisted and the inferior branch of the retinal artery was seen to be narrowed. Kurz described another case, that of a woman aged 33 who suddenly saw a cloud in front of her right eye. The arteries were much narrowed, but vision and the retina became normal in 24 hours. Abadie[94] (1935) referred to a girl aged 18 who suddenly lost vision in the left eye. Sometime later a similar event occurred in the right eye. She entered a school for the blind and learned Braille. Several months later vision was partly restored in the left eye. Four years later vision returned almost to normal within 15 minutes.

In this group blindness was associated with epilepsy, jaundice, diplopia, blowing the nose, cold sensitivity, menstruation, malaria, paroxysmal hypertension, and retinal tuberculosis. Other cases were similar to those of Group 2, but the descriptions were so brief that they could not justifiably be placed in that group. It is remarkable that in several cases the blindness was relieved by amyl nitrite.

### CLINICAL PICTURE

It is unlikely that the clinical picture as presented in my few cases is a fully characteristic one. However, the pattern of symptoms and signs has not varied a great deal from case to case, and this suggests that at least the basic features are representative. All but one of the patients in this study were males, but from a knowledge of the incidence of atherosclerosis it is safe to predict that females will not prove so immune. Usually the patient is elderly, but occlusion of the internal carotid artery may develop in persons with premature atherosclerosis as early as the 30's or 40's. Intermittent blindness in one eye may be the first and only prodromal symptom, or it may alternate with transient attacks of contralateral paralysis and paresthesia. Episodic aphasia also occurs. The attacks of blindness seem not to occur at the same time as the other neurological symptoms. After one, two, or more months of intermittent blindness a contralateral hemiplegia is precipitated, and this, while often severe, is not invariably so. Usually the attacks of blindness cease with the onset of the stroke, but in Case 3 symptoms were occurring four or five times a week one year later. In Andrell's Case 7, periodic blindness did not begin until after the cerebrovascular accident, and in Case 6 of the present series two attacks occurred about seven years after the first neurological symptoms. The significance of the various time relations will be discussed later under "Mechanism." It should be emphasized here that in my experience the patient will not volunteer the information about blindness after the stroke, possibly because he is ill or handicapped or forgetful.

94. Abadie, C.: Du spasme des artères centrales de la rétine, Presse méd. **33**:1026, 1935.

The attacks of blindness are probably always unilateral, but one patient claimed that at times both eyes were affected. The blindness most commonly comes on as though a blind were being lowered or raised, and vision returns from the opposite direction. In Case 2 the blindness was described as "a fog rolling in." Scintillating phenomena and many of the accurately described, but bizarre, visual symptoms recorded in the literature as accompanying fleeting blindness will probably be duplicated in this syndrome, but so far they have not been encountered. During the phase of recovery, vision was distinctly blue in the cases of Wagenmann and Weiss. The attacks last from a minute or so up to seven minutes or more. They may occur with almost any frequency, from once a month to once or twice a day. The onset, as well as the recovery, may be sudden or gradual. Usually blindness is total at the height of the attack, and the pupil no doubt dilates, but this has not been seen personally.

There is no accompanying dizziness or light headedness, such as occurs when many healthy people straighten up after bending over. In none of the present cases could a precipitating factor be determined, such as exertion, bending, eating, use of the eyes, or standing erect. If the cases presented by Zentmayer, Harbridge, Traquair, and Sanders are examples of carotid disease, then bending and exertion may be expected to bring on attacks. In cases of bilateral carotid occlusion exertion is prone to bring on transient amaurosis.

A satisfactory ophthalmoscopic examination has not been made in a proved case of occlusion of the internal carotid artery during an attack of blindness, but it is almost certain that the picture often described in the literature will be found, namely, paleness of the optic disk, attenuated, bloodless arteries, and perhaps the "cattle-truck" appearance of the blood in the veins. From a consideration of the many cases of transient blindness in the literature in which only field defects were complained of, it is to be expected that similar partial blindness will occur in cases of this syndrome, one branch of the central retinal artery being affected before the others; or, indeed, the other vessels may not become involved at all. In Case 2 optic nerve atrophy was found several months after the stroke, but this occurrence apparently is unusual.

Since the visual symptoms seem related in some way to occlusion of the internal carotid artery, a few details concerning that process must be included. The pathologic basis for the arterial occlusion is atherosclerosis. The occlusion is usually localized in the region of or within the carotid bulb, just distal to the origin of the internal carotid artery in the neck. It is a fact that atherosclerosis occurs in that site just as early and just as severely as in the abdominal aorta. Since the substratum of carotid occlusion is atherosclerosis, evidence of the process elsewhere is often present, for example, angina pectoris, cardiac infarction, intermittent claudication, absence of the peripheral pulses, or gangrene of the lower extremities. The blood pressure is usually normal, or perhaps the systolic reading is slightly elevated. Syphilis seems to play no part. In many cases virtual occlusion of the carotid bulb takes place without producing symptoms, such as hemiplegia; this occurrence raises the possibility that visual symptoms may sometimes be coincidental with loss of the carotid pulse, rather than causally related.

Whereas occlusion most frequently occurs within the carotid bulb, obliteration of the lumen of the carotid artery has also been shown to take place distal to the origin of the ophthalmic artery; and while such a case, characterized by transient

blindness, has not been encountered, the discovery of such an arrangement would obviously be of great value in deducing the mechanism of the transient blindness.

The diagnosis depends a great deal on keeping the syndrome in mind. The absence of pulsation in the internal carotid artery may prove diagnostic, but at times the pulse in the external carotid artery is accentuated, making it impossible to detect absence of pulsation in the internal carotid artery. In a few cases the external carotid artery was found at autopsy to be greatly increased in size. Palpation must be carried out as high up in the neck as possible, to get above the stem of the external carotid artery, which soon divides into its several branches. The expansile thrust of the internal carotid artery should be gently sought for. Since the carotid bulb is a focus for atherosclerosis ulceration and thrombotic deposits rough pressure should not be used. In a few cases the external carotid artery is also occluded, and then absence of the pulse in the temporal artery suggests the diagnosis. Occasionally, anomalies of the carotid arteries occur, and among these is absence of one internal carotid artery. Only once have I heard a bruit at the carotid bifurcation, so that auscultation is not a very helpful procedure. A decrease in the pressure within the central retinal artery on the affected side was mentioned by Krayenbühl and Weber,[95] but in none of the present cases was careful examination made for such a change. A rough estimate has been made by pressing on the eyeball with the index finger until the blood flow ceases in the central retinal artery, but it has been difficult to decide whether the results are significantly altered from the normal. A difference in the surface temperature of the cheek and that of the forehead has not been found. Carotid arteriography is the only way by which to make a positive diagnosis in life. I feel, however, that the procedure is not entirely safe in the presence of extensive disease of the carotid artery and that its routine use to confirm a clinical impression is not justified, especially after a cerebrovascular disaster has occurred.

### DIAGNOSIS AND TREATMENT

It is not unlikely that when intermittent blindness serves as the herald of an impending hemiplegia the ophthalmologist will be the first consulted. There will then arise the important questions: Is a cerebrovascular accident to be expected? If so, can any preventive measures be taken? I cannot answer these questions from actual personal experience, as I have never been fortunate enough to examine a patient before the onset of the stroke. In regard to the first question, one should first try to rule out or in each of the diagnoses listed in the historical summary of transient blindness. As has been already pointed out, detailed follow-up reports of the cases in the literature are almost completely lacking, so that the relation of many reported cases to the present syndrome can only be surmised. A history of migraine in a young patient would justify placing the case in the migraine group, but as unilateral headache may accompany carotid disease, "migraine" in an older patient should be held suspect. The presence of definite hypertension, especially if the intermittent visual disturbance involves only a sector of the visual field, points to involvement of the central retinal artery (Case 7), rather than to carotid occlusion. The possibility of polyarteritis nodosa

95. Krayenbühl, H., and Weber, G.: Die Thrombose der Arteria carotis interna und ihre Beziehung zur Endangiitis obliterans v. Winiwarter-Buerger, Helvet. med. acta **11**:289, 1944.

or its close relative temporal arteritis must be kept in mind, fever, the presence of tender, painful temporal arteries, the evidence of multiple foci of disease, and the raised sedimentation rate suggesting the diagnosis. Bilateral periodic blindness is unlikely to be due to unilateral carotid disease, but bilateral carotid disease may be characterized by bilateral disturbance of vision, coming on during exercise. Attacks of so-called "reflex amaurosis" should be quite apparent from the history.

There still remains to be differentiated the large number of cases of "idiopathic transient blindness." Since the case reports in the literature usually do not include the blood pressure levels and the details of the general health of the patient, no reliable diagnostic rules can be laid down. Such cases tend to fall in the younger age groups, but carotid disease can be present in the 30's or 40's, even in women. Tentatively, I should feel that good pulsation in the internal carotid arteries is strong evidence against a threatening stroke; but, as will be seen in the section on "mechanism," there are so many unknowns that formulation of any rule is not justified.

The second question—Can any preventive measures be taken if a diagnosis of carotid disease is suspected at a stage prior to the cerebrovascular accident?—is also to be answered without personal experience. If the stroke results from thrombosis of the internal carotid artery or of a large collateral channel within the circle of Willis, it would seem logical to use anticoagulants, and even autonomic-blocking agents, to assist in postponing vascular occlusion and to enhance further collateral circulation. The length of time that such a regimen would have to be maintained is, of course, unknown. If the main obstruction should prove to be in the internal carotid artery, as seems highly likely, one could speculate that a surgical anastomosis of the external and internal carotid arteries might be an effective therapeutic measure. To refer again to the question of carotid arteriography, it might be said that the procedure is justified as an investigative measure, remembering, however, that the vision in one eye will be further jeopardized. If the patient is seen when fleeting attacks of blindness, paralysis, paresthesias, or aphasia herald the onset of a stroke, arteriography may prove necessary before a rational therapeutic approach can be worked out.

## MECHANISM BY WHICH BLINDNESS IS PRODUCED

A complete and exact explanation of the mechanism responsible for the intermittent ischemia of the retina cannot be offered at present. However, a rather large number of clinical observations has been assembled over a long period, and when these are considered in an orderly fashion, the search for the correct mechanism is greatly narrowed. After a brief review of the practical anatomy of the circulation of the eye, the clinical observations alluded to will be presented.

The ophthalmic artery arises from the internal carotid artery as that vessel lies on the medial side of the anterior clinoid process, and near the apex of the convexity, directed forward. It enters the orbital cavity through the optic foramen, lying lateral to and below the optic nerve. The central retinal artery arises from the ophthalmic, near the optic foramen, and runs forward in or outside the dural sheath of the nerve. The central retinal artery arises singly or, more commonly, as a common trunk with one of the ciliary arteries. It is about 280 $\mu$ in diameter at its origin, but as it pierces the dura it gives off a few branches and is reduced to about 180 $\mu$ in diameter. These branches are important, for

they run parallel with the parent vessel and are the nutrient vessels of the intraorbital portion of the optic nerve. The central retinal artery does not give off any branches in its intraneural portion. It divides on or just beneath the surface of the optic disk into its superior and inferior branches, each of which, in turn, divides into nasal and temporal branches. There is said to be no anastomosis between any of the four retinal arteries, but an insignificant anastomosis may be present on the optic disk between small branches of the retinal arteries and branches from the circle of Zinn. Thus, the central retinal artery has no effective collateral system of supply, so that, as far as is known, obstruction of this vessel should always lead to severe or complete retinal ischemia.

The internal carotid artery, from which arises the ophthalmic artery, originates at the bifurcation of the common carotid artery in the upper part of the neck. At its origin it expands slightly to form the carotid bulb; then, passing directly upward, it traverses the temporal bone, passes through the cavernous sinus, and about 2.0 cm. distal to the origin of the ophthalmic artery it divides into the anterior and middle cerebral arteries. The external carotid artery, the other terminal branch of the common carotid artery, soon dwindles in size as it gives origin to the superior thyroid, lingual, occipital, and facial branches.

In contrast to the dearth of collaterals of the central retinal artery, the ophthalmic artery and its branches make abundant and effective anastomotic connections with branches of the external carotid artery of the same and of the opposite side. This rich collateral supply has been recognized by anatomists for decades, but only a few clinicians have investigated it. Elschnig [96] (1893) carried out nine postmortem injection experiments, and in all it could easily be shown that an abundant collateral circulation existed between the branches of the external carotid and the ophthalmic arteries of the same and opposite sides. Walsh and King [97] (1942) repeated these experiments, using colored solutions and radiopaque material, with the same results; but the number of experiments and the pressures used were not mentioned. I have been unable to confirm these results, using injections of isotonic sodium chloride solution, but further trials are necessary before making a conclusion contrary to such a well-established fact. Adson [98] (1942), in the surgical treatment of carotid artery-cavernous sinus aneurysm with pulsating exophthalmos, placed a silver clip on the ophthalmic artery in three cases without damage to vision. However, it is well known that such aneurysms open up collateral channels to an extraordinary degree. Reference will be made later to this problem of the collateral circulation of the ophthalmic artery.

Although it has not been possible for me to examine the fundus at the height of an attack of blindness, it is probably safe to assume that the retinal vessels are ischemic or bloodless, in whole or in part. Ophthalmoscopic examination during or throughout an episode of temporary obscuration of vision has been recorded by at least 30 observers; and, while the details may vary, the general

96. Elschnig, A.: Über den Einfluss des Verschlusses der Arteria ophthalmica und der Carotis auf das Sehorgan, von Graefes Arch. Ophth. **39**:151, 1893.

97. Walsh, F. B., and King, A. B.: Ocular Signs of Intracranial Saccular Aneurysms; Experimental Work on Collateral Circulation Through Ophthalmic Artery, Arch. Ophth. **27**:1 (Jan.) 1942.

98. Adson, A. W.: Surgical Treatment of Vascular Diseases Altering Function of Eyes, Am. J. Ophth. **25**:824, 1942.

picture is rather constant. There is usually pronounced retinal pallor, associated with bloodless, thread-like arteries in one or more sectors. (It is not clear whether the choroidal circulation is interrupted.) During this period, vision is lost, and the pupil dilates. With the restoration of the circulation, vision returns. Assuming that interruption of the retinal blood supply is undoubtedly the immediate cause of the transient amaurosis, the site and nature of the obscuration may now be considered.

My first six cases of intermittent blindness were characterized by occlusion of the ipsilateral internal carotid artery and by contralateral hemiplegia. Elementary consideration of this clinical picture would justify the conclusion that the intermittent symptoms, including the blindness, were due to intermittent constriction of a severely diseased internal carotid artery, causing temporary interruption of the blood flow to the eye and to the brain. However, there is abundant evidence against such a mechanism:

1. In cases characterized by both intermittent blindness and transient paralysis or paresthesia, the blindness seems never to occur at the same time as the other symptoms. If transient occlusion of the internal carotid artery were responsible, the ischemia of the retina and the brain should be simultaneous, if not every time, at least some times.

2. With the onset of the hemiplegia, vision is not usually affected, whereas one would expect it to be if gradual occlusion of the internal carotid artery were the cause of the stroke and the preceding attacks of blindness. In Case 2 blindness of the ipsilateral eye was found three months after the onset of the hemiplegia, but this appears to be the exception. Yet the event which determines the attacks of blindness bears some relation to the cause of neurologic deficit, for after the onset of the stroke intermittent blindness usually ceases. Nevertheless there are exceptions to this rule, too, for in Case 3 periodic blindness was still occurring approximately four times a week, one year after the onset of aphasia and right hemiplegia. In Case 7 reported by Andrell [2] periodic attacks of blindness first appeared after the onset of a stroke. In Case 6 of the present series attacks of blindness first appeared seven years after the first neurologic signs. Thus, there are already several exceptions to the rule that temporary blindness precedes and ceases with the stroke, and only further observations will clarify this point.

3. In addition to the foregoing clinical observations, a series of facts indicates strongly that obstruction of the internal carotid artery is not directly responsible for the ischemia of either the retina or the brain. Ligation of the internal carotid artery for infraclinoid and supraclinoid vascular lesions has now been carried out many times without affecting vision in the ipsilateral eye. Schorstein [99] collected detailed accounts of 49 cases in which the internal carotid artery was ligated in the neck. No visual disturbances were recorded, although neurologic complications occurred in 22.4% of the cases. Olivecrona [100] (1944) reported 24 similar cases, and visual deficit was not mentioned. Adson [98] (1942), in treating pulsating exophthalmos, ligated the carotid vessels in the cervical region and the internal

99. Schorstein, J.: Carotid Ligation in Saccular Intracranial Aneurysms, Brit. J. Surg. **28**:50, 1940-1941.

100. Olivecrona, H.: Ligature of the Cartoid Artery in Intracranial Aneurysms, Acta chir. scandinav. **91**:353, 1944.

carotid artery as it emerged above the cavernous sinus and placed a silver clip on the ophthalmic artery in three cases without damage to vision. All this is good evidence that the ophthalmic artery has an abundant anastomosis with other vessels, especially the external carotid artery. One might anticipate, therefore, that ligation of the common carotid artery would prove more injurious to vision, since flow in the external carotid artery would be interrupted also; yet there is much evidence to the contrary. Siegrist [101] (1900) surveyed the reports of 997 cases in which the common carotid artery was ligated for various reasons and concluded that in relatively healthy patients ligation of the common carotid artery has never led to any visual disturbances whatsoever. Schorstein [99] collected 11 cases of similar ligations without ill effects. Elschnig [96] compressed the carotid artery on many occasions without producing a visual change. Ligation in animals is also unattended by visual disturbance.[101] Evidence is thus overwhelming that rather abrupt occlusion of the carotid arteries does not significantly alter the blood supply to the eye, although intraocular tension may fall. It is highly unlikely, then, that slow occlusion by atherosclerosis, with or without superimposed thrombosis, would lead to symptoms, visual or cerebral, in the vast majority of cases. That intermittent blindness can occur when the internal carotid artery is already blocked is proved by Andrell's Case 7, in which attacks of blindness continued after the onset of the stroke, when arteriography showed complete obstruction of the internal carotid artery. It would be most useful in such a case to know whether the vessel is blocked only in the region of the bulb or whether the internal carotid artery in its entire course is filled with clot. If the vessel were so filled, the thrombus might be blocking the mouth of the ophthalmic artery, or even extending along it. In Case 1 of the present series the internal carotid artery above the bulb was observed to be empty post mortem, but one cannot say with certainty that a clot was not there originally and then gradually softened and disappeared, without being organized. In most cases of carotid occlusion associated with hemiplegia, when pathologic examination is made soon after the stroke, the internal carotid artery above the bulb is found to be filled with antemortem blood clot, which extends upward past the ophthalmic artery to the anterior and middle cerebral arteries. Unfortunately, in none of these cases in which I have made examination has intermittent blindness occurred. It would be interesting to have contrast dye injected into the carotid artery above the occluded bulb, in such a case as Andrell's. Evidence that the external carotid artery is not to be incriminated, at least in all cases, was furnished by my Case 3. The internal carotid artery was occluded clinically; yet compression of the common carotid artery failed to precipitate blindness, although the patient was having spontaneous attacks almost daily. In Andrell's Case 5, in which there was monocular blindness and contralateral hemiplegia, the ophthalmoscopic picture of thrombosis of the central retinal artery was presented. Arteriography showed occlusion of the internal carotid artery, but the external carotid artery was well filled, indicating that the blindness was not due to lack of major collaterals. One would have to postulate that in such a case thrombosis or embolism had blocked the origin or the course of the central retinal artery. This clinical picture of permanent monocular blindness, optic

101. Siegrist, A.: Die Gefahren der Ligatur der grossen Halsschlagadern, für das Auge und das Leben des Menschen, von Graefe's Arch. ophth. **50**:511, 1900.

nerve atrophy, and contralateral hemiplegia has been described for 75 years as typical of thrombosis of the internal carotid artery, but persistent blindness is the exception rather than the rule.

Thus, many facts contradict the proposition that the intermittent blindness, and even the cerebral symptoms, are directly due to intermittent obstruction of the internal carotid artery. Another possibility is that the immediate cause of these symptoms lies in the gradual occlusion of large anastomotic channels, such as the anterior or posterior communicating arteries, the internal carotid having previously been occluded. This could well explain the cerebral symptoms but would not account for the attacks of blindness, provided that the collateral supply to the ophthalmic artery is as good as one is led to believe. It would still be difficult to explain why intermittent cerebral symptoms do not take place at the same time as the visual disturbance.

The physiologic concept which seems best to fit the clinical picture is vasospasm. It is likely that contraction of the smooth muscle of a vessel wall can effectively interrupt blood flow, as exemplified in Raynaud's syndrome and the aura of migraine. Certainly, vasospastic occlusion would be compatible with the transiency and the reversibility of the attacks of blindness. Nerve fibers have never been demonstrated on the retinal vessels; however, there is much indirect, but nonetheless good, evidence that they are present. If the conclusion, stated above, is valid, i. e., that collateral blood flow via the ophthalmic artery is sufficient to supply the eye when the internal carotid artery and the proximal portion of the ophthalmic artery are obstructed, vasospasm must involve the central retinal artery and/or its branches. Benson's [40] patient had attacks sometimes affecting the whole field of vision, but most frequently implicating only one section. On examination during one attack, the inferior temporal artery of the retina was entirely bloodless for about 4 disk diameters, while the other vessels seemed normal, vasospasm involving only one branch. That a sector defect can be associated with occlusion of the internal carotid artery is suggested by the case of Foerster and Guttman, in which selective blindness occurred repeatedly in the lower field of the right eye. A later operative note mentioned that the internal carotid artery on that side scarcely pulsated. In cases in which total blindness occurs the central retinal artery is probably involved, rather than its four branches separately. In support of the role of vasospasm is the tendency, reported by several observers, for glyceryl trinitrate or amyl nitrite to prevent or alleviate attacks. That vasospasm should persist as a rule for only a few minutes finds no parallel or analogy in any other vascular process, except perhaps migrainous aura. An important question is: Why do transient symptoms tend to appear at a time when some vessel is so nearly occluded that the passing of a few weeks or months is sufficient to result in a disastrous hemiplegia? If vasospasm does occur, it must be closely related to the threatened occlusion of that vessel, be it the internal carotid or a communicating artery.

Why vasospasm should show itself for long periods only in the central retinal artery is not clear. Perhaps it depends on anatomical factors or intraocular dynamics still to be elucidated. Moniz, Lima, and de Lacerda,[102] who first described spontaneous occlusion of the internal carotid artery, attributed the fleeting paresthesias

102. Moniz, E.; Lima, A., and de Lacerda, R.: Par Thrombose de la carotide interne, Presse méd. **45**:977, 1937.

and paralyses to reflex vasomotor impulses arising in the internal carotid artery. Similar impulses could affect either the direct or the collateral vessels to the retina. Hultquist [103] also mentioned the possibility of nerve impulses arising in the carotid sinus. Adams [104] suggested that the blindness depended on spasm of the branches of the external carotid artery, which is supplying the retina owing to lack of flow via the internal carotid artery. This is in keeping with the generally accepted belief that the extracranial vessels undergo vasomotor changes more readily than intracranial ones. Several cases have been reported in which bending, stooping, gazing downward, or exercising has brought on attacks, observations which are evidence for mechanical factors rather than vasospasm. Further speculation is not warranted at present, and it must be emphasized that the role of vasoconstriction in these cases can be defined in the future only if careful clinical and arteriographic studies are followed later by detailed postmortem examination, gross and microscopic, of the carotid arteries, the mouth and the entire course of the ophthalmic artery, the mouth and the entire length of the central retinal artery, and the proximal portion of its branches. The circle of Willis must also be studied. Only when the anatomical substratum of the syndrome is known can the functional aspects be elucidated with any degree of reliability.

The idea of vasospasm, far from being new, was offered as early as 1864 by Hughlings Jackson to explain an attack of blindness lasting five minutes in one of his patients. He used the term "retinal epilepsy." However, the concept of embolism of the central retinal artery continued to be strongly favored, in spite of views to the contrary expressed by Loring, Nettleship, Smith, and Ramorino. The last-named author attributed his cases of transient blindness to vasospasm, while the others held that insufficiency of the general circulation or thrombosis of the central retinal artery was responsible. Zentmayer thought that pressure on the optic nerve by sclerosed internal carotid arteries could cause transient amaurosis. As the rarity of embolism and the relative frequency of thrombosis of the central retinal artery became established, transitory episodes were attributed with increasing frequency to vasospasm or vasoconstriction. Whether or not a vessel has first to be diseased by virtue of atherosclerosis, syphilis, a toxic substance, or arteritis has remained a point of contention, the consensus being, however, that such a disease process must be present. There is, of course, no good proof for such a view, for pathologic studies have seldom, if ever, been made.

Before this section on mechanism is concluded, the phenomenon of recurrent field defects warrants further discussion. Case 7 has been included in this paper to illustrate this complaint. The patient, a woman aged 47, had repeated attacks of blindness in the lower half of the visual field of the left eye. Several similar cases have been reported previously, a permanent defect usually being precipitated sooner or later. The process has been watched ophthalmoscopically and does not differ from that seen in transient total blindness, except for the number of vessels involved. The vasoconstrictive or ischemic phenomenon may involve the upper or lower retinal arteries, and sometimes one nasal or one temporal branch is affected separately. That there is a special predilection to involvement of the upper portion

103. Hultquist, G. T.: Über Thrombose und Embolie der Arteria carotis und hierbei vorkommende Gehirnstörungen, Jena, Gustav Fischer, 1942.

104. Adams, R. D.: Personal communication to the author.

of the retina was noted by Minton,[105] who in a follow-up study of 54 cases of occlusion of the central retinal artery and its branches found the superior arteries more frequently affected, and the temporal branch oftener than the nasal. This relative sparing of the nasal portion of the retina had been noted by Coats [106] and was attributed by him to a better collateral anastomosis on the nasal side. The order of susceptibility to ischemia would appear to be as follows: superior temporal, superior nasal, and, finally, the inferior half. My Case 3, in which blindness always appeared in the lower nasal field and then moved diagonally upward until the upper temporal field became blind, is in agreement with such an order of susceptibility. However, there are many exceptions to this rule, which will have to be tested by further observations.

A more reliable dictum indicates that in partial vascular insufficiency of the retina the visual field defect is a horizontal one, while ischemia of the visual system more centrally produces a vertical defect, usually a homonymous hemianopsia. Priestley Smith [7] was apparently the first to point this out, and Uhthoff [107] (1925) later concluded that thrombosis of the central retinal artery invariably produced an upper or a lower field defect, never a medial or a lateral one. The importance of this rule in the diagnosis of transient phenomena is obvious. Case 7, in which only the lower half of the visual field was involved, illustrates this principle.

Thrombosis of the central retinal artery has an important bearing on the problem of intermittent blindness, for Uhthoff found prodromal attacks of obscuration of vision, either total or in part of one visual field, in 30% of his 86 cases. Thus, in the older age groups transient blindness may indicate disease of either the central retinal artery or the internal carotid artery. There is no certain way of distinguishing these two forms; but if it is assumed that atherosclerosis of the central retinal artery is the basis of later thrombosis in that vessel, there may be general clues. A personal study of the distribution of atherosclerosis in normotensive and hypertensive patients has led to the expectation that atherosclerotic plaques should appear in the central retinal artery only, or almost only, in the presence of hypertension. A review of my own limited clinical material has shown hypertension to be present in every instance of occlusion of the central retinal artery. Minton,[105] in his follow-up study, recorded the blood pressure in some 23 cases of occlusion of the central retinal artery. Only 15 could be considered cases of uncomplicated thrombosis, and of these, high blood pressure was present in 13. Thus, there is evidence that thrombosis of the central retinal artery is a complication of hypertension. Case 7 of the present series was the only one in which definite hypertension was found, and the carotid pulse was strong when the patient was first seen. In such cases an atherosclerotic plaque may narrow the central retinal artery sufficiently so that relatively little further vasoconstriction will occlude the lumen. On the other hand, peripheral vasospasm may have been largely responsible, for the attacks had been occurring for 5 or 10 years, and atherosclerosis should have altered in that length of time.

105. Minton, J.: A Clinical Study of 54 Cases of Occlusion of the Central Artery of the Retina and Its Branches, Proc. Roy. Soc. Med. **30**:285, 1936.

106. Coats, G.: Obstruction of the Central Artery of the Retina, Roy. London Ophth. Hosp. Rep. **16**:262, 1905.

107. Uhthoff, W.: Periodische Verdunkelungen der Augen, Klin. Monatsbl. Augenh. **75**: 469, 1925.

In brief, transient monocular blindness, occurring in older patients in the presence of hypertension, probably heralds the threatened occlusion of the central retinal artery, especially if pulsation in the internal carotid artery is good. Any subsequent hemiplegia is not directly related and means that a separate intracranial vessel has been occluded. Such a clinical picture is to be distinguished from that presented by the first six cases, in which occlusion of the internal carotid artery seems to be the fundamental process, and in which transient blindness and the onset of a hemiplegia are closely related. At the same time, it must be remembered that since occlusion of the internal carotid artery undoubtedly occurs without hemiplegia, it may well happen, on occasion, that intermittent blindness is the only manifestation of such occlusion.

## SUMMARY

A new syndrome is described in which transient monocular blindness precedes or is associated with contralateral hemiplegia. The clinical picture is attributed to occlusion of the cervical portion of the internal carotid artery on the side of the affected eye. Six cases are presented, in one of which the diagnosis was confirmed at necropsy, and in another by arteriography, while in the others the diagnosis was made on clinical grounds.

The clinical picture is characterized by repeated brief episodes of blindness in one eye. Usually in the course of weeks or months, a stroke occurs, after which the attacks of blindness cease. However, exceptions to these rules are known. Pulsation in the internal carotid artery is typically absent. The patients usually are elderly and show clinical evidence of atherosclerosis in other vessels of the body. The high incidence of carotid disease is pointed out. The difficulty in deciding the cause of intermittent blindness is mentioned.

The problem of treatment awaits further study and the accumulation of more case reports.

The intermittent blindness probably depends on the interruption of the blood flow to the retina. The mechanism by which this occurs is discussed at some length, with the conclusion that vasospasm or vasoconstriction must be incriminated. The exact relation of the carotid occlusion to the transient phenomena is far from clear.

A detailed summary of the literature relating to blindness due to intermittent retinal ischemia is presented.

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# EFFECTS OF ULTRASONIC WAVES ON THE REFRACTIVE MEDIA OF THE EYE

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A PROGRAM of experimental research has been undertaken in cooperation with the Department of Physics at the Catholic University of America to study the production and possible clearing of opacities in the refractive media of the eye by means of ultrasonic irradiation. Although up to the present it has not been possible to clear such opacities, significant findings have been obtained in the study of the effects of intense ultrasonic waves in excised calves' eyes. It is the purpose of this paper to report this work at its present stage of development.

The term "ultrasonics" designates sound waves having a frequency beyond the audible range, i. e., above 30,000 cps.

Although ultrasonic waves differ from ordinary audible sound only by having a higher frequency and smaller wave length, these properties greatly influence their propagation through matter. Much information about the behavior of different media has been obtained using ultrasonic waves as a tool.[1] The ability of a substance to transmit ultrasonic waves depends on such properties as the density elasticity, viscosity, and structure of the medium. High-energy ultrasonic beams bring about many thermal, mechanical, and even chemical changes in the medium. This, naturally, has led many to seek to develop the use of ultrasonics in therapy and diagnosis.

In Europe,[2] ultrasonics has been widely used in the treatment of a great variety of diseases. "Good results have been reported, but unfortunately most of the therapeutic evaluations have been based on clinical impressions alone and few controlled

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From St. Elizabeths Hospital (Drs. Lavine and Langenstrass); the National Institute of Health (Dr. C. M. Bowyer), and the Catholic University of America (Dr. F. E. Fox, Dr. V. Griffing, and Dr. W. Thaler).

1. Bergmann, L.: Ultrasonics and Their Scientific and Technical Applications, Edition 3, Ann Arbor, Mich., Edward Bros., 1942.

2. Badtke, G.: Ultraschallbehandlung der Auges, Klin. Monatsbl. Augenh. **114**:193-196, 1949; Nuovo Cimento, Atti del Convegno Internazionale di Ultracustica, Rome, July 14-17, 1950, Supp. 2, Vol. 7, Ser. 9, Sect. 4 and 5, Bologna, Nicola Fanichelli, Editore, 1951.

observations have been made. As a result, very little accurate scientific information on the therapeutic effectiveness of ultrasound has been obtained."[3] As previously stated by one of us (F. E. F.),[4] "There is need of fundamental work on simpler systems, [i. e. systems simpler than the complicated living organism] to determine first the various mechanisms by which ultrasonic waves produce such effects," before experiments in biology and medicine can really be interpreted.

For example, one needs to distinguish between primary effects of ultrasonic waves, such as instantaneous particle velocity, pressure, temperature, and density changes in the sound wave itself, and secondary effects, such as an increase in local temperature due to absorption. There is great need for the careful specification of the physical variables of the experiment, so that, for example, the frequency and intensity[5] dependence are determined. Only when these are known can the experiments be correlated with the fundamental knowledge of the physicist working in ultrasonics.

Very little experimental work has been undertaken to determine the effects of ultrasonics on the eye. The first experimental work on this subject was reported by Zeiss[6] in 1938.

Zeiss studied the effects of ultrasonic waves of high intensity with a frequency of about 250 kc. on the vitreous and lens of eyes taken from recently slaughtered 2- to 3-year-old cattle. He used both plane waves and focused beams of ultrasonic waves. Most of the significant results were developed by the focused beam, for which he estimated an intensity of 10 watts per square centimeter, but did not say how it was obtained. Zeiss distinguished three phenomena which it produced. 1. The vitreous was irreversibly liquefied in two- to four-seconds' exposure to the ultrasonic beam. 2. Furthermore, a zonularis-like opacity, composed of very fine bubbles (two-to-eight minutes' irradiation) was produced. This opacity developed in the periphery of the embryonic nucleus ("perinuclear cataract"). 3. Another type of lenticular opacity was observed after longer exposure. This opacity consisted of leucoma-like white spots in the outer cortical layers. Apparently, these white spots developed only where the lens had been in contact with the test tube in which it was supported. The zonularis type of opacity disappeared in from 5 to 30 minutes after removal of the lens from the ultrasonic beam. The second type of opacity, located directly beneath the capsule, constituted an apparently permanent, i. e., irreversible, change. Zeiss ascribed the first type of opacity to cavitation.[7] He offered no explanation for the second type.

More recently, Kawamoto[8] has reported results of irradiating excised lenses and vitreous bodies of adult white rabbits and guinea pigs with high-intensity plane ultrasonic waves of 547-kc. frequency. A few human lenses were also used in these

3. Ultrasonics in Diagnosis, Editorial, J. A. M. A. **146**:1033 (July 14) 1951.

4. Fox, F. E.: The 1950 International Ultrasonics Congress at Rome, July 14-17, 1950, J. Acous. Soc. Am. **23**:233-234, 1951.

5. That is, energy passing through 1 sq. cm. per second.

6. Zeiss, E.: Über Linsenveränderungen an herausgenommenen Rinderlinsen durch Ultraschalleinwirkung, von Graefes Arch. Ophth. **139**:301-322, 1938.

7. Cavitation is the production of small, gas-filled cavities in the sound-irradiated substance, as a result of alternating changes in pressure and temperature of the ultrasonic wave.

8. Kawamoto, I.: Experimental Studies on the Effect of Ultrasonic Waves on the Eyeball, Nippon Gankwa Gakukwai Zasshi **51**:12-16 (Feb.) 1947. We are dependent on the translator for the accuracy of the report.

experiments. In all cases, the vitreous body was liquefied after short exposure. In irradiation of the lens, the deep layers of the cortex surrounding the embryonic nucleus became turbid. This phenomenon, Kawamoto identified with the "ultrasonic cataract" of Zeiss.[6] On longer exposure to the ultrasonic beam, a spotted, dense turbidity developed around the nucleus. In addition, a small, thin, cloudy area of turbidity developed within the cortex under the capsule. In agreement with Zeiss, Kawamoto observed that the major portion of the spume-like turbidity disappeared after a short time; however, the spotted and thin cloudy turbidity did not disappear.

Histologic study by Kawamoto of irradiated rabbit eyes showed disruption of lens fibers, scattering of the walls, and abnormal agglutination of the protoplasm, which showed no changes with staining. There were also sporadic nonstructural degenerative foci adjoining the edge of the embryonic capsule, which stained dark red with Van Gieson's solution. These foci showed two or three air bubbles. On the basis of these findings, he concluded that the turbidity was caused by cavitation. The spotty and thin cloudy turbidity was irreversible. It appeared in front of the cavitation on the side of the lens facing the quartz crystal. This opacity appeared denser when the temperature at the posterior capsule measured about 55 C. The author reproduced this opacity by immersing the lens in an isotonic sodium chloride solution at the same temperature, i. e., 55 C. Kawamoto concluded that the turbidity in these irradiated lenses may have developed because of the heat, but he maintained an attitude of doubt.

In 1949 Badtke reported a few experiments on live rabbits in which no pathological changes were observed beyond a slight transient opacity and swelling of the superficial corneal layers. This change had disappeared completely when the eyes were enucleated, four days later. On the basis of these experiments, Badtke concluded that it was safe and justifiable to "irradiate human eyes."

## PRESENT EXPERIMENTS

Since much good work is difficult to interpret or to reproduce because of the author's failure to specify the precise acoustic conditions under which the experiments were made, one of the first objectives of our work was to determine such variables as exactly as possible. Whenever possible, the acoustic intensity and frequency, the temperature reached by the absorbing medium during irradiation, and other factors, have been determined and recorded.

In order to facilitate these measurements, we have until now worked exclusively with excised eyes. The eyes were obtained from freshly slaughtered calves, 4 to 6 weeks old, and were kept in a water-ice mixture at 0 C. until immediately before use. After the eyes had been in this cold mixture for several hours, fluffy perinuclear opacities were observed in the lenses. These delicate zonular opacities cleared up, starting from the outside, as the eyes were observed at room temperature. Up to the time of this report, about 600 eyes have been used in our experiments, and each observation has been checked many times.

Ultrasonic energy—much like visible light—can be applied to the eye in the form of a parallel beam of plane waves. If a parallel beam is applied to the lens of the intact eye, there will be fairly uniformly distributed concussion of all structures in the path of this beam. Fairly uniform irradiation of the cornea, the lens, and the vitreous body, as well as of the retina, choroid, and sclera, occurs. No selective

irradiation of the lens is possible in this case. When, however, the parallel ultrasonic beam falls first upon a concave reflector and is thus focused, the situation becomes quite different. Previous experiments [9] had shown that nearly all the energy in a high-frequency ultrasonic beam could be concentrated by a concave reflector in a very small area, where the intensity would be very large. This, therefore, seems to be an ideal way of investigating the effect of ultrasonic energy on the different media and tunics of the eye. If such a convergent ultrasonic beam is focused in the substance of the crystalline lens, one obtains an almost completely selective effect upon the lens fibers. The effect on the cornea, retina, and other structures of the eye becomes very small, in fact, negligible. An experiment can easily be arranged so that the energy focused in 1 cu. mm. of tissue inside the lens will traverse the cornea with an energy density of only one-hundredth that value. Furthermore, considerable intensities can be produced without all the experimental difficulties inherent in maintaining the very high voltages required to produce the same intensities with plane waves. Focusing, however, rapidly becomes less effective when the wave length approaches the dimensions of the reflector. This happens as the frequency falls below 1 megacycle (mc.). For these reasons, most of our experiments have been made with sharply focused beams having a frequency greater than 1 mc.

TABLE 1.—*Crystal Data*

| Frequency | Shape | Dimensions | Area of Back Electrode |
|---|---|---|---|
| A 3.25 mc. | Rectangle | 2.5 × 3.5 cm. | 1.8 × 1.8 cm. |
| B 2.00 mc. | Circular | 3.8 cm. diameter | 1.8 cm. diameter |
| C 1.00 mc. | Square | 5.1 × 5.1 cm. | 3.5 × 3.5 cm. |
| D 500 kc. | Circular | 2.5 cm. diameter | 1.8 cm. diameter |

## EXPERIMENTAL DETAILS

All measurements were made with X-cut quartz crystals as transducers. Each crystal was mounted in the center of one end of a small water-filled tank. The tank measured 8 by 8 by 15 in. (20.3 by 20.3 by 38 cm.). Four different crystals were used, with the following fundamental frequencies: 500 kc., 1.00 mc., 2.00 mc., and 3.25 mc. Other data for these crystals are given in Table 1. The grounded side of the crystal was in contact with the water in the tank; the high-voltage face of the crystal was exposed to the air. The driving voltage was supplied by various radio-frequency oscillators. These oscillators were capable of maintaining a potential of about 1,500 volts across the crystal for the frequencies above 1 megacycle (mc.). Their range extended up to 2,000 volts for the 500-kc. crystal.

The experimental arrangement is shown in Figure 1. The reflector used was a watch glass 7.5 cm. in diameter and having a radius of curvature of 6.4 cm. The holder for the lens and that for the whole eye can be moved in three dimensions, so that any part of the eye or lens can be brought to the point at which the ultrasonic beam is focused. Figure 2*A* and *B* show the holders in detail. The lens holder contains three channels for insertion of a thermocouple into the various regions of the lens. The holder for the whole eye is so arranged that the cornea is always gently pressed against the front of the holder by a light spring. The whole system can easily be removed from the tank for insertion of lens, thermocouples, etc., without changing the relative positions of the reflector and holders. It is then easily replaced at a marked position across the

9. Fox, F. E., and Griffing, V.: Experimental Investigation of Ultrasonic Intensity Gain in Water Due to Concave Reflectors, J. Acous. Soc. Am. **21**:352-354, 1949. Griffing, V., and Fox, F. E.: Theory of Ultrasonic Intensity Gain Due to Concave Reflectors, ibid. **21**:348-351 (July) 1949.

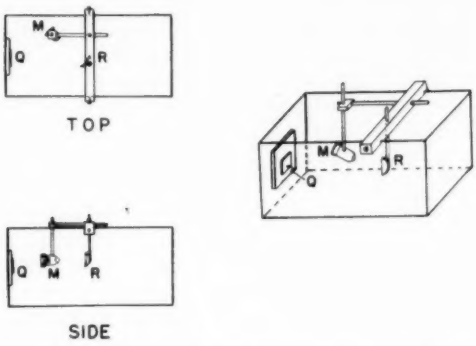


Fig. 1.—Experimental arrangement. *Q* indicates quartz crystal; *M*, holder for eye or lens; *R*, reflector.

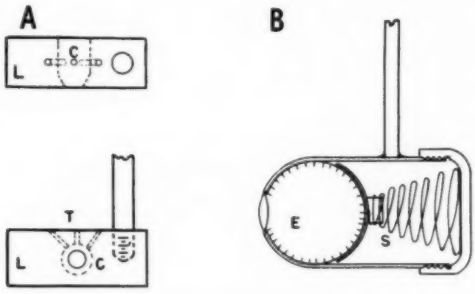


Fig. 2.—Details of holders for lens (*A*) and holder for whole eye (*B*). *L* indicates lucite® block; *C*, cavity for holding lens; *T*, channels for thermocouple; *S*, spring for holding whole eye, *E*, in position.

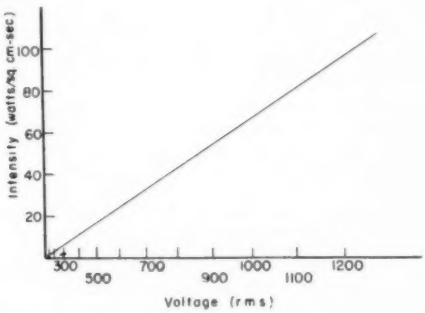


Fig. 3.—Acoustic intensity in the focal region as a function of the voltage driving the crystal. The frequency equals 3.25 mc.

top of the tank. In this way, it was possible to reproduce accurately the experimental conditions for the irradiation of any chosen part of the eye in successive exposures.

Other holders were used for exposure of the desired parts of the eye to plane progressive ultrasonic waves and to plane standing waves of very high intensity. These holders will be described when these experiments are reported in detail.

Several methods were used to measure the intensity of the applied ultrasonic beam. The most direct method was to measure the gain [9] of the ultrasonic-source-reflector system at a given frequency. The gain was defined as the ratio of the intensity (maximum or average) in the focused region to the intensity in the plane ultrasonic beam. This determination was made by measuring the pressure exerted on a very small glass bead in the focal region. This enables [9] one to calculate the energy density and intensity in the focal region. In a similar manner, the energy density of the plane wave at the crystal was determined, in this case using a larger obstacle. The energy density in the plane wave as it leaves the transducer was also calculated [9] from the voltage on the crystal, its known sharpness of resonance, and the resonance frequency. Since the intensity varies as the square of the voltage applied to the crystal, a series of readings of intensity as a function of applied voltage was made. In Figure 3 the intensity is plotted as a function of $V$. This calibration curve was used thereafter to determine either the intensity of the plane ultrasonic wave or that in the focal region. After the importance of the role of local heating had been recognized, attention was focused more upon measurements of temperature than on those of intensity.

In the experiments with plane standing waves, no attempt was made to determine accurately the absolute intensities used. The applied voltages were used to estimate relative intensity, and even absolute intensities with fair accuracy in certain geometrical containers.

Measurements of the local temperatures in various irradiated regions were made by means of a very small thermocouple (copper-constantan wire, Brown & Sharpe No. 30). Preliminary experiments were made to insure that the effect of the ultrasonic waves impinging directly upon the thermocouple was negligible, i. e., less than 0.5 C. The cool junction was kept in a water-ice mixture. The thermocouple was calibrated by comparison with a mercury-in-glass thermometer whenever it was used, since it was more convenient to use a microammeter than a potentiometer for observation of the temperature, which at times was changing rapidly.

### EXPERIMENTS AT 3.25-MC. FREQUENCY WITH FOCUSED BEAM

*Continuous Irradiation.*—Since the experimental observations were repeated many times (usually more than five times for each observation), a typical result will be described for each experiment. All experiments made to insure correct positioning of focus in the eye and the lens are omitted. Obviously, many such tests were necessary before the experiments described were possible.

The first experiments were made with whole calves' eyes. The experimental arrangement was such that the eye could be observed during irradiation. With 1,000 volts on the crystal (1,000 volts rms) there developed in one second a white opacity (Lavine cataract [10]) within the lens at the focus of the ultrasonic beam. After 30 seconds this opacity had grown so that the whole lens became a dense, firm, white mass. At lower voltages (and correspondingly lower intensities) the time necessary for the appearance and growth of this white opacity increased, and the final product was less dense. A threshold voltage was finally attained below which no opacity was seen, even though the irradiation was continued for as long as 30 minutes. In each instance the Lavine opacities thus produced were irreversible. They showed no tendency to change after the end of the irradiation. Under these conditions no changes could be observed in any other part of the eye except for moderate increase in local temperature. If the eye was placed so that the cornea was at the focus, a somewhat similar opacity developed through the

10. In order to distinguish this type of irreversible ultrasonic opacity from those described by Zeiss and Kawamoto, we shall refer hereafter to this opacity as the Lavine cataract.

whole thickness of the cornea. However, the voltage threshold was higher. Many observations were also made on the excised lens supported in the holder, shown in Figure 2*A*. Table 2 gives the exposure times necessary for the lens opacity in both the whole eye and the excised lens to become just dense enough to be seen in cross illumination.

*Intermittent Irradiation.*—One of the first questions to be investigated was whether or not the Lavine cataract represents a direct result of the ultrasonic irradiation itself (and as such is dependent directly on the ultrasonic intensity, that is, the alternating pressure, temperature, and density changes in the sound wave) or whether it is a secondary effect. The most probable secondary effect would be local heating of the medium due to absorption of the ultrasonic energy concentrated at the focus of the convergent ultrasonic beam. If the effect were direct, one

TABLE 2.—*Exposure Time Necessary for Formation of Lens Opacity with Focused Beam* and Continuous Irradiation*

| Voltage | Time of Formation (Sec.)† | Voltage | Time of Formation (Sec.)† |
|---|---|---|---|
| Lens in Excised Whole Eye | | Lens Only (Excised) | |
| 1,000 | 2 | 650 | 3.9 |
| 710 | 6 | 650 | 4.5 |
| 710 | 5 | 650 | 4.1 |
| 600 | 14 | 600 | 5.2 |
| 600 | 8 | 600 | 4.9 |
| 500 | 60 | 600 | 5.2 |
| 500 | 78 | 500 | 10.5 |
| 500 | 68 | 500 | 11.0 |
| 500 | 47 | 500 | 11.0 |
| 500 | 61 | 450 | 35.0 |
| 500 | 57 | 400 | 46.0 |
| 500 | 42 | 400 | 46.5 |
| 450 | 111 | 400 | 45.5 |
| 425 | 119 | | |
| 400 | 300 (no opacity) | | |

* Frequency, 3.25 mc.
† Time from beginning of irradiation to first visible appearance of the opacity.

would expect that opacification would occur before the critical temperature level was reached. One would also expect that the time of irradiation necessary for the production of a just observable opacity at a given intensity would be more or less independent of whether the irradiation was continuous or intermittent. On the other hand, if the effect were secondary, such as the coagulation of proteins that one observes in the heating of egg albumin, the interruption of the irradiation before coagulation starts would result in a certain amount of cooling. A longer total irradiation time would be necessary at a given intensity, and the threshold intensity would be strongly dependent upon the length of the on-off periods during irradiation. A series of such intermittent irradiations was made on both the excised lens and the lens in the whole eye. The results are given in Table 3. For an intensity well above the minimal intensity necessary to produce a Lavine cataract in 10 seconds, no opacity can be produced at all if the on-off ratio is lower than 1 : 1. This suggested strongly that the effect is indeed a secondary one, probably local heating.

*Thermocouple Measurements.*—In order to test this hypothesis, holes were drilled into the lens holder (as shown in Figure 2*A*), so that a very small thermocouple could be inserted and held in place in the lens at a point where the ultrasonic beam was focused. Difficulty was encountered at first. Although large increases in local temperature were recorded, the opacity no longer formed in the ultrasonic focus but formed somewhat to the front of this region, and thus not in contact with the junction. With use of even smaller thermocouple wire and care that a cavity was not left in the lens substance around the thermocouple junction (which happens if the junction moves slightly after it is inserted in the lens) this difficulty was eliminated. It was found that the opacity became visible only when the local temperature in the lens was higher than 68 C. Within a wide range of intensities

TABLE 3.—*Exposure Time Necessary for Formation of Lens Opacity with Focused Beam and Intermittent Irradiation*

| Voltage | On-Period (Sec.) | Off-Period | Total Time On-Off Period (Sec.) | Results |
|---|---|---|---|---|
| | | Lens in Excised Whole Eye | | |
| 600 | 6 | 60 | 270 | No opacity |
| 600 | 6 | 6 | 54 | No opacity |
| 600 | 6 | 3 | 42 | One very faint opacity after fifth on-period |
| 600 | 6 | 2 | 22 | One opacity at end of third on-period |
| 600 | 6 | 1 | 17 | One opacity at 3 sec. of third on-period |
| 600 | 3 | 1 | 11 | One opacity at end of third on-period |
| 600 | 3 | 1 | 17 | One opacity at 1 sec. of fifth on-period |
| 500 | 3 | 3 | 300 | One very faint opacity at end of run; 300 sec. total time |
| | | Lens Only (Excised) | | |
| 500 | 3 | 1 | 26 | One opacity at 2 sec. of 7th on-period |
| 500 | 3 | 1 | 26 | One opacity at 2 sec. of 7th on-period |
| 500 | 3 | 1 | 28 | One opacity appeared during 7th off-period |
| 500 | 3 | 2 | 42 | One opacity at 2 sec. of 9th on-period |
| 500 | 3 | 2 | 40 | One opacity at end of 8th off-period |
| 500 | 3 | 2 | 51.5 | One opacity at 1.5 sec. of 11th on-period |

above the minimal value, it was possible to determine the time at which the opacity just appeared by noting the time at which the thermocouple current reached a given value, i. e., that the local temperature reached that level necessary for coagulation to occur. Another type of observation confirms the decisive role of local temperature. With continuous irradiation the intensity was increased in small steps—two minutes being allowed for the local temperatures to reach a steady state at any one intensity. A corresponding rise in the steady-state temperature for each step was observed until the temperature reached the threshold value. At this temperature the Lavine opacity was observed to develop. Figure 4 shows the result of several such experiments. The temperature is plotted as the ordinate. The points give the steady-state temperature attained after the voltages are set at the values indicated on the abscissa. The minimal temperature for opacity production is seen to be about 70 C.

In Figure 5 the temperature is plotted as a function of time for an experiment in which a constant voltage (320 volts) was applied for 175 seconds and then turned off. A barely discernible opacity was observed in this case.

*Gross and Microscopic Description of the Lavine Cataract* (Focused Beam Type).—The Lavine cataract assumes a variety of geometrical shapes (Figs. 6 and 7). If the focal point lies within the lens, it is fusiform in its initial stage. The poles

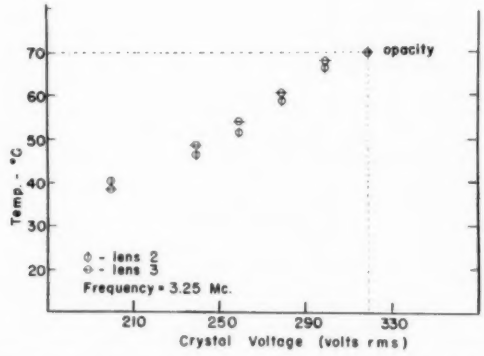


Fig. 4.—Temperature developed in the lens in the focal region of the ultrasonic beam as a function of applied voltage. All observations were made at the end of a two-minute irradiation period.

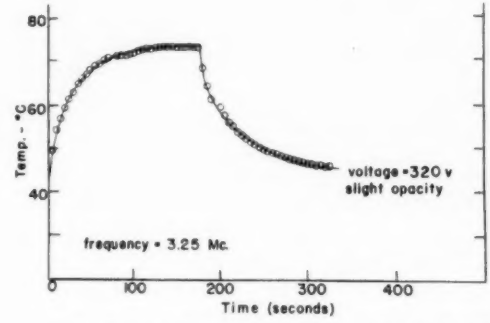


Fig. 5.—Temperature developed in the lens in the focal region of the ultrasonic beam as a function of time with a constant applied voltage.

of the spindle are slightly rounded. Its long axis coincides with the axial ray of the ultrasonic-convergent-wave front. The denseness and size of the opacity depend upon the intensity, frequency, and time of irradiation. The fusiform area is entirely opaque, firm, even hard. Prolonged or high-intensity irradiation changes the fusiform shape. The poles now become markedly rounded. Gradually, with continued irradiation, the opacity tends to involve the entire lens. This process is a function of the temperature and time of application.

Examination with the slit-lamp microscope showed a somewhat uneven, shaggy or fuzzy surface everywhere along the interface between fusiform opacity and the surrounding clear lens. The opaque area appeared uniformly dense throughout.

Equatorial frozen sections at 10-$\mu$ thickness and unstained were studied microscopically at two levels of magnification (100 and 440 $\times$).

Corresponding to the grossly recognized dense, whitish opacity, there was a circumscribed, approximately circular area of a uniform, dark, grayish-brown color. Here the lens stroma appeared very dense, apparently swollen. It seemed to have undergone a kind of granular disintegration. Outlines of a few lens fibers here and there were indistinctly seen, especially with higher magnification and reduced sub-

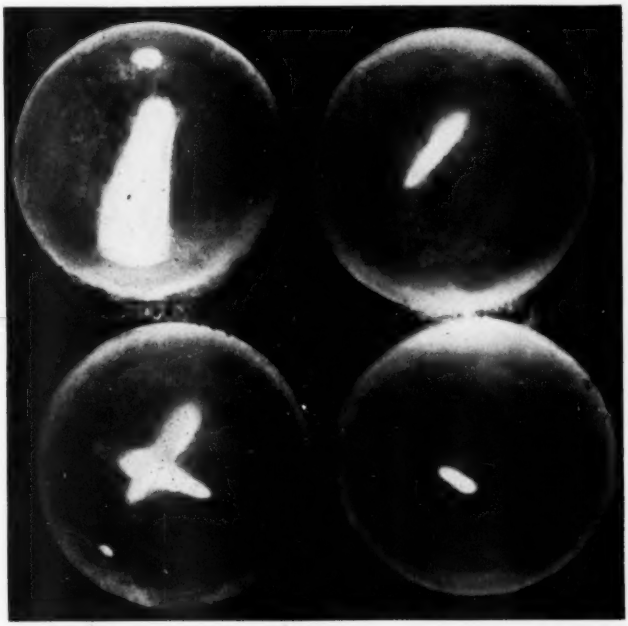

Fig. 6.—Lavine cataract produced with focused ultrasonic radiation. Frequency ($f$) equals 3.25 mc.; the time of irradiation of the lenses shown ranged from less than 1 second for the smallest opacity, to 10 seconds, for the largest opacity. All voltages were 800 volts rms. The lens with the three cataracts (two crossed) indicates how a cataract can be placed in almost any desired area within the lens.

stage light. The finer architecture of the individual fibers was completely invisible and seemed to have been destroyed to a very great extent.

This central area of disintegration and discoloration was surrounded by a narrow, concentric ring of brown discoloration. Structural changes were only slight in this zone. They were seen only where the zone came in contact with the central area of apparently complete disintegration.

A second, broader concentric zone surrounded the first narrow, dark-brown ring. Here the lens stroma presented a uniform, pale-brown color with no trace

of structural change or damage. Farther outward even the brown color was no longer visible. The lens fibers appeared healthy, transparent, and clear.

*Additional Changes.*—When the anterior or posterior pole of the Lavine cataract came in contact with the lens capsule, continued irradiation led to local rupture of the capsule. This rupture could often be heard as a clear-cut "puff." One saw this small rupture with the slit-lamp microscope as an irregular elevation of the polar region of the dense opacity and a small area of capsular laceration. The cataract then projected slightly into the capsular gap.

As long as the lens capsule remained intact, in none of the above experiments did we observe any definite signs of cavitation or opacities, such as were described

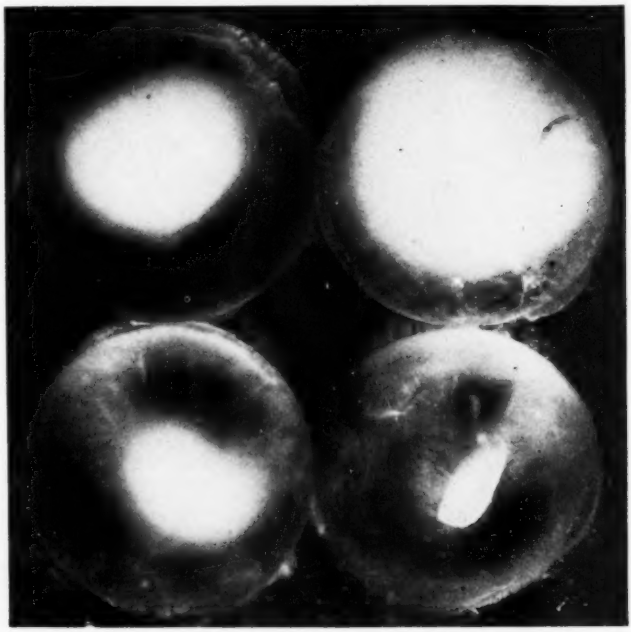

Fig. 7.—Lavine cataracts: The three diffuse cataracts were produced by irradiation of the whole eye with plane waves for 3 to 10 minutes. A Lavine cataract produced by the focused beam is included for comparison. Frequency was 3.25 mc.; 1,200 volts rms.

by Zeiss. This is because in a given medium at higher frequencies the intensity must be increased before cavitation occurs. Occasionally one would observe a few small, isolated bubbles in the lens after it had been irradiated at high intensity for short periods (so as to keep the temperature below the threshold value for production of the Lavine cataract). But it was found that such observations were always associated with rupture of the lens capsule. These bubbles are explained as follows: There is probably little dissolved gas in the lens substance. After the capsule has been ruptured, the sound wave can carry gas nuclei from the water tank into the

lens substance. Since cavitation occurs at much lower intensities in media containing gas nuclei, small bubbles form when the capsule is ruptured.

### EXPERIMENTS AT 2 AND 1 MC.

Similar experiments were then carried out with crystals B (frequency 2.00 mc.) and C (frequency 1.00 mc.), with the same focusing apparatus. The results were in all cases similar to those obtained at a frequency of 3.25 mc. However, at the frequency of 1 mc. larger numbers of cavitation bubbles were observed in lenses with ruptured capsules or with absence of the capsule. In such cases the Lavine cataract was difficult to obtain, presumably because of the more rapid flow of heat from the lens.

We also verified the observations of Zeiss on the liquefaction of the vitreous humor by ultrasonic waves. Our experiments, however, were carried out at a frequency of 2 mc. and a voltage of 1,450.

### FORMATION OF HEAT OPACITIES IN A THERMOSTATIC REGULATED BATH

To determine whether elevation of temperature alone is sufficient to cause opacities to develop in the refractive media of the eye, the following series of experiments was performed. A heater, stirrer, thermometer, and thermostat were arranged in a 1-gal. (3.8 liter) Dewar flask, and the temperature was allowed to come to equilibrium at temperatures approximately 1 C. apart. The lens and portions of the capsule and cornea were then placed in the constant temperature bath for periods varying from 5 to 30 minutes. In all cases there was a temperature threshold above which an opacity appeared: for the capsule, about 55 C.; for the lens in the capsule, 63 C.; for the cornea, 50 C. In particular, in this experiment the lens opacity developed from the outside of the lens and grew inward. When the temperature of the bath was significantly high above the threshold value, lenses with opaque, dense outer regions and less dense central regions were obtained. The rate of coagulation increased with the temperature of the bath, as did also the density of the final opacity. Those opacities that were formed in a bath at 70 C. were rather hard and dense in five or six minutes. But a 30-minute period was necessary before a lens at 63 C. became completely filled with a light cloudy opacity.

The observed minimal temperature for the ultrasonic production of the Lavine cataract lies above that obtained in the temperature bath. This is to be expected, since the rate of formation of the opacity is so slow at the minimal temperature that a higher temperature is attained before the opacity is macroscopically observable. In the constant temperature bath the lens could be held at the minimal temperature for an indefinitely long time.

### IRRADIATION WITH PLANE WAVES

The thermocouple and controlled-temperature experiments had shown why preliminary experiments with plane waves had failed to produce any observable opacities. That is, the heat produced through absorption was dissipated by conduction and convection to the water in the tank. The steady-state elevation of temperature of any point in the irradiated eye (or lens) did not reach the threshold value for coagulation. The following experiments were undertaken with parallel waves, in which the heat losses were minimized and the sound intensity was increased: The whole eye was placed in the holder shown in Figure 2*B*, and held less than 1 cm.

in front of the 3.25-mc. crystal. The maximum voltage obtainable (1,200 volts) was used to drive the crystal. In two minutes a Lavine cataract started forming at or near the geometric center of the lens, growing outward until the whole lens became opaque after long-continued irradiation. It must be realized that the whole eye in this irradiation was in a much more intense sound field than would have existed in the beam of plane progressive waves. This is because multiple reflections inside the holder form an irregular standing-wave system. At the same time, the lens is prevented from losing its heat by the surrounding ocular tissue. The tunics of the eye insulate it from the water in the tank. There was lens opacity, but none on

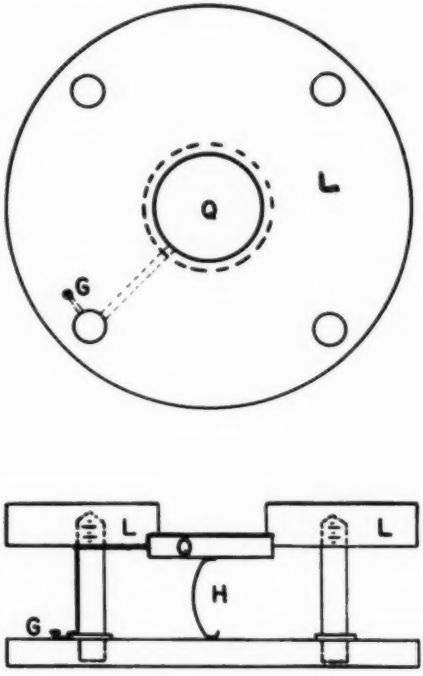


Fig. 8.—Experimental arrangement for use with 500-kc. plane waves. *Q* indicates quartz crystal; *L*, lucite® disk; *G*, grounded electrode; *H*, high-voltage electrode.

the cornea, presumably because the cornea was kept cool by contact with the water in the tank.

The above experiment was repeated, the excised lens only being used. The time necessary to produce the cataract in this case was three minutes.

The eye was next placed in a holder with an open back and with a sound-absorbing-diffusing disk several inches beyond the eye, so that no standing-wave system could be formed. After irradiation for three minutes with the 1,200-volt beam, the lens was found to have a Lavine cataract which was densest at a point somewhat in front of the geometric center of the lens and had obviously started forming there.

This is because the more intense radiation is absorbed in the part first traversed by the beam; the absorption itself removed energy from the beam on its way through the viscous lens medium. In the excised lens, an equivalent opacity was produced after six to eight minutes in a plane progressive wave of the same intensity.

Finally, two eyes were placed with their corneas together and irradiated in the holder shown in Figure *2B* for 10 minutes at 1,200 volts. Upon removal of the eyes from the holder, the parts of the cornea in contact with each other were found to be opaque. Only a slight opacity was discernible in each of the two lenses. These opacities were on the sides facing the corneas. In one lens, this was on the side of the lens receiving the less intense radiation.

It should be noted that the time required to produce equivalent opacities with the focused beam not only is very short (one second), but is the same for both the excised lens and the lens in the whole eye. With plane waves, either progressive or standing, the time needed to produce an opacity is much longer. Furthermore, the

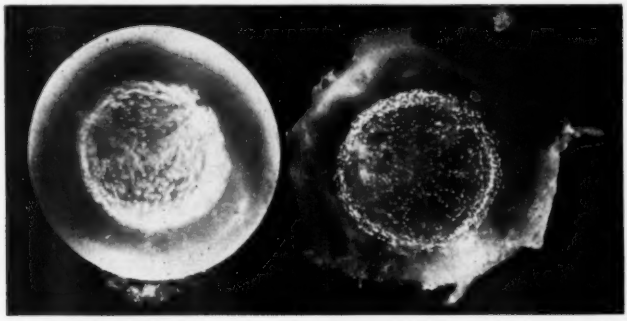

Fig. 9.—Zeiss cataracts: opacities produced by irradiation for two minutes with 500-kc. plane wave; 2,000 volts rms. The capsule has been removed from one lens, after irradiation, in order to show the detailed structure of the Zeiss cataract.

time necessary to produce the opacity in the excised lens is six to eight minutes for plane progressive waves, as compared with three minutes when the lens is irradiated in the whole eye.

These experiments confirm the conclusion that the Lavine cataract is related to the equilibrium-temperature distribution during irradiation and therefore to the geometry and to such properties of the medium as conductivity and viscosity.

In none of the above experiments was there any marked cavitation, or production of reversible opacities due to clouds of cavitation bubbles, such as were described by Zeiss and Kawamoto. Since cavitation occurs more readily at the lower frequencies, at which Zeiss and Kawamoto worked, it was decided to attempt to reproduce their observations. It is believed that it is in some such phenomenon as cavitation that one might hope to produce a clearing of either the naturally occurring cataract or the irreversible, Lavine cataract.

A 500-kc. quartz crystal was mounted under a hole drilled in a slab of lucite,® as shown in Figure 8. The hole in the lucite® was filled with water, and excised

lenses were placed directly on the crystal in this container. A potential of 2,000 volts was applied to the crystal.

In this arrangement the reflection of the ultrasonic waves from the top water-surface sets up a standing-wave system which is constantly fluctuating, but in which the intensity is increased several times over that which one would have had with a progressive-wave system. Upon irradiation for periods of two minutes, the Zeiss perinuclear opacities were obtained, obviously being due to cavitation bubbles in the lens. These opacities were reversible, although even after periods of as long as an hour there were enough remaining bubbles to outline the region in which they had progressive-wave system. Upon irradiation for periods of two minutes, the Zeiss cataracts. In these first irradiations no attempt was made to keep the temperature of the water below that necessary to produce the irreversible opacities which were observed at higher frequencies. As a result, one occasionally obtained small regions of dense white opacities, such as those described by Zeiss, and particularly by Kawamoto. These always occurred where the lens was in contact with the crystal or the lucite® holder. However, as soon as we adopted the procedure of continually flushing out the lens container with cool water during irradiation, these opacities failed to develop. This did not in any way hinder the formation of the Zeiss, or fine-cavitation-bubble opacity. It seems that the irreversible opacities described by Zeiss and Kawamoto were of the same type as we had observed in these experiments and were of the same nature as those we produced so rapidly during irradiation at higher frequencies; i. e., they are due to local heating.

## COMMENT

This series of ultrasonic experiments on the eye were undertaken with the hope that ultrasonic waves, when properly used, would provide a means for the disruption and ultimate liquefaction of the opaque lens-protein and lens-fiber systems of clinical cataracts. This hypothesis was based upon (*a*) many reports that large molecules can be broken into smaller ones by means of ultrasonic waves; (*b*) the dispersal and coagulation of aerosols by means of ultrasonic waves; (*c*) cases of the spontaneous disappearance of the opaqueness in the traumatic cataract of youth and in cataracta hypermatura fluida.[11]

We have succeeded in producing new types of ultrasonic cataracts which in some respects are similar to certain clinical cataracts. The focused beam type can be produced at specified areas, within a few seconds, without any observable effect on the rest of the eye. Thus, we have a readily available material and technique for experimental cataract research.

The perinuclear opacities observed by Zeiss and Kawamoto have been reproduced. These are due to cavitation. They are reversible and suggest the only mechanism as yet observed which may prove useful in cataract clearing. The results of these investigations suggest two extensions of these experiments for further research in determining whether cataracts can be cleared by ultrasonic waves. First, the effects of cavitation on the crystalline lens should be further studied. The unusual mechanical and chemical effects that are known to accompany cavitation

11. Berens, C.: The Eye and Its Diseases, Edition 2, Philadelphia, Saunders Company, 1950. Duke-Elder, W. S.: Textbook of Ophthalmology, Vol. III: Diseases of the Inner Eye, St. Louis, C. V. Mosby Company, 1941.

may provide means of achieving this goal. Second, the effects of intermittent high-frequency, high-intensity irradiation should be fully investigated. In this way, it may be possible to produce cavitation without concomitant heating at high frequencies, where the obvious advantages of focusing may be fully utilized.

It must be emphasized that all these observations concern excised eyes. The effects of ultrasonic irradiation on the living eye should be studied by exactly controlled experiments in which the physical variables are specified as carefully as possible. One might expect that ultrasonic irradiation may influence the permeability of the lens capsule, with consequent improvement in the nutrition of the lens.

## CONCLUSIONS

1. The formation of fine lenticular perinuclear opacities is the result of cavitation. These are produced by irradiation with low-frequency, high-intensity ultrasonic beams. This type of opacification is reversible.

2. The formation of the Lavine cataract is due to the absorption of high-frequency, high-intensity ultrasonic radiation, which results in high local temperatures. This type of opacity is irreversible.

3. The liquefaction of the vitreous produced by ultrasonic irradiation is irreversible.

## SUMMARY

A series of ultrasonic experiments have been performed on calves' eyes, with the use of frequencies of 0.5, 1, 2, and 3.25 mc. and of various intensities. Corneal opacities, vitreous liquefaction, and cataractogenic effects have been produced with plane progressive, plane standing, and focused ultrasonic beams. The fine perinuclear cavitation and spotted cortical opacities, as well as the vitreous changes observed by Zeiss and Kawamoto, have been seen in our laboratory. A new type of experimental cataract and the mechanism for its production are described.

# SURGICAL TREATMENT OF ESOTROPIA WITH BILATERAL ELEVATION IN ADDUCTION

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IN A PREVIOUS paper,[1] it was shown that (1) in certain cases of horizontal squint the vertical deviations can be secondary to dissociation of the eyes; (2) examination of the eyes in the straight-up and straight-down positions of gaze is of great diagnostic importance, and (3) cases of horizontal squint with a vertical component can be classified into four main groups: Group I: esotropia with bilateral elevation in adduction; Group II: esotropia with bilateral depression in adduction; Group III: exotropia with bilateral elevation in adduction; Group IV: exotropia with bilateral depression in adduction.

The essential diagnostic findings in Group I include an esotropia that is noncomitant in that the squint is greater for near and downward gaze than for distance and upward gaze. Right hypertropia is found on gaze to the left, and left hypertropia, on gaze to the right. The convergence near point is very good. The delayed cover test usually shows some type of dissociated vertical divergence. The elevation in adduction may be difficult to demonstrate bilaterally, since many of these patients have marked dominance of one eye and others have crossed fixation.[2] In such cases the patient should be forced to fix with the abducted eye by occluding the other eye. When the fixing eye is in extreme abduction, uncovering the adducted eye will demonstrate the elevation.

The purpose of this paper is to discuss the results of surgical intervention in cases of Group I, demonstrating corrections obtained by (*a*) bilateral recession of the medial rectus, (*b*) bilateral resection of the lateral rectus, (*c*) resection and recession of one eye, and (*d*) recession of the inferior oblique combined with operation on the horizontal muscles.

## BILATERAL RECESSION OF THE MEDIAL RECTUS

CASE 1.—R. I., aged 3 years, came to the Infirmary in 1946, with a history of crossed eyes since the age of 2 years, following measles.

With atropine cycloplegia, refraction was as follows:

O. D.: + 3.50 sph. ⊂ + 1.25 cyl., ax 90 = 20/100
O. S.: + 3.50 sph. ⊂ + 1.50 cyl., ax 90 = 20/100

The results of examination are shown in Figure 1*A*.

On Feb. 17, 1947, a bilateral 4-mm. recession of the medial rectus was done.

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From the Department of Ophthalmology, Illinois Eye and Ear Infirmary, of the University of Illinois College of Medicine.

1. Urist, M. J.: Horizontal Squint with Secondary Vertical Deviations, A. M. A. Arch. Ophth. **46**:245 (Sept.) 1951.

2. Chandler, P. A.: Operations in Convergent Squint, Am. J. Ophth. **34**:375, 1951.

Fig. 1 (Case 1).—(Feb. 4, 1947) *A*, before surgical correction: (*a*) 35 degrees of right esotropia, with right hypertropia for near without glasses; (*b*) 25 degrees of right esotropia, with right hypertropia for near with glasses; (*c*) 20 degrees of right esotropia looking up; (*d*) marked elevation of left eye in adduction; (*e*) marked elevation of right eye in adduction; (*f*) 35 degrees of esotropia looking down.

*B*, postoperative appearance following a bilateral 4-mm. recession of the medial recti: (*a*) 10 degrees of right esotropia for near with glasses two months after operation; (*b*) 25 degrees of right esotropia for near without glasses two months after operation; (*c*) eyes straight for near with glasses eight months after operation stereopsis; (*d*) elevation of left eye in adduction still present; (*e*) elevation of right eye in adduction still present; (*f*) 20 degrees of right esotropia for near without glasses; (*g*) eyes straight looking up; (*h*) 30 degrees of left esotropia looking down; (*i*) convergence near point after operation, 25 mm.

*C*, four years after operation: (*a*) eyes straight for near with glasses, third-grade fusion; (*b*) eyes straight for near without glasses; (*c*) convergence near point, 25 mm.; (*d*) and (*e*) eyes level on lateral gaze, elevation in adduction gone; (*f*) eyes straight looking up; (*g*) eyes straight looking down.

The results of postoperative examination are shown in figure 1*B* and *C*.

The bilateral recession straightened the eyes in all positions with elimination of the elevation in adduction. It corrected 20 degrees of esotropia on looking up and 35 degrees of esotropia looking down. The bilateral recession of the medial rectus did not reduce the convergence near point. The immediate result of surgical intervention was a marked undercorrection (Fig. 1*B*, *a*, *b*). A second operation was scheduled two months after the first, but, owing to fortuitous

Fig. 2. (Case 2).—*A*, preoperative examination: (*a*) 35 degrees of right esotropia for near without glasses; (*b*) 25 degrees of left esotropia for distance without glasses; (*c*) convergence near point, 80 mm., can be 20 mm. when patient concentrates; (*d*) 20 degrees of left esotropia for near with glasses; (*e*) 20 degrees of left esotropia for distance without glasses; (*f*) and (*g*) marked bilateral elevation in adduction.

*B*, one year after a bilateral 3-mm. recession of the medial recti: (*a*) eyes straight for near with glasses; (*b*) 5 degrees of left esotropia for near without glasses; (*c*) eyes straight for distance without glasses; (*d*) elevation of left eye in adduction almost gone; (*e*) elevation of right eye in adduction almost gone; (*f*) convergence near point, 25 mm.; (*g*) eyes straight looking up; (*h*) eyes straight looking down; (*i*) 10 degrees of left esotropia looking down.

circumstances, it was delayed eight months. When the patient was examined, eight months after operation, his eyes were straight with glasses. Screening showed only esophoria with glasses, and he had third-grade fusion. The improvement in this patient has been steady and, as figure 1*C*, *a* and *b* shows, four years after surgical correction, his eyes are straight with and without glasses. The correction has been reduced to +2.00 sph. ⊂ +1.50 cyl., ax 90 for each eye, with 20/20 vision.

CASE 2.—G. B., aged 5 years, came to the Infirmary in 1948, with a history of crossed eyes since the age of 18 months, following measles. A brother, two years younger, has a squint.

With atropine cycloplegia, refraction was as follows:

O. D.: +4.00 sph. ⊂ +2.25 cyl., ax 90 = 20/30
O. S.: +4.25 sph. ⊂ +2.50 cyl., ax 90 = 20/30

The results of examination are shown in Figure 2*A*.

On June 20, 1949, a bilateral 3-mm. recession of the medial rectus was done.

The results of postoperative examination (Sept. 14, 1950) are shown in Figure 2*B*.

Although the measurements of the prism-cover test did not indicate much convergence excess, the finding of more esotropia on looking down than up indicated that an overaction of the medial recti existed. This was confirmed by the postoperative results. The bilateral 3-mm. recession corrected 20 degrees of esotropia on looking up and 35 degrees of esotropia on looking down. The convergence near point following operation was 25-mm. The eyes were straight for distance with glasses (*c*). There was 5 degrees of left esotropia for near without glasses (*b*). The eyes could be straight looking down (*h*), but when convergence impulses became hyperactive, 10 degrees of estropia appeared on looking down (*i*). The bilateral elevation in adduction was greatly reduced, although the screening measurements of the hypertropia had not diminished very much as yet.

CASE 3.—A. G., aged 12, came to the Infirmary in 1949, with a history of a crossed left eye since the age of 3 years.

With atropine cycloplegia, refraction was as follows:

O. D.: +4.00 sph. ⊂ +1.25 cyl., ax 105 = 20/20
O. S.: +4.50 sph. ⊂ +1.50 cyl., ax 90 = 20/30

The results of examination are shown in Figure 3*A*.

On Feb. 11, 1950, a bilateral 5-mm. recession of the medial rectus was done.

The results of postoperative examination (July 25, 1950) are shown in Figure 3*B*.

Fusion was second grade at zero. Retinal correspondence was normal; the patient saw a cross with the after-image test.

The etiologic factor in the overaction of the medial recti in this case was that of an accommodative squint. The size of the pupils, studied by photographs under constant lighting conditions, helped to demonstrate this. When the accommodation was relaxed, the pupils were large, and the eyes could be straight with glasses (Fig. 3*A*, *a*), or converge 5 degrees without glasses (Fig. 3*A*, *c*). When the eyes were convergent 20 degrees, the pupils were much smaller (Fig. 3*A*, *b*). Before surgical correction the squint varied from 10 to 30 degrees of esotropia with or without glasses; at no time could the eyes be found to be straight when the patient was examined in the clinic. A bilateral recession of 3 or 4 mm. was contemplated to eliminate the overaction of the medial recti, but, because of such large screening measurements, 80 △ looking down and 65△ for near, a bilateral 5-mm. recession of the medial rectus was done.

Immediately after surgical intervention there was a tendency toward divergence, and the convergence near point was reduced to 110 mm. In this case, although large screening measurements for near and looking down were present before operation, the eyes were straight with photographs and under surgical anesthesia. For this reason a bilateral 3.5-mm. recession would most likely have been better and would not have affected the convergence near point so adversely. The recession corrected 15 degrees of esotropia on looking up and 25 degrees on looking down (50 △ up and 80 △ down with the cover-test measurements). The convergence near point gradually improved to 50 mm. The elevation in adduction was much improved, although the left hypertropia was still present. The left hypertropia, which was found only with screening, and the greater elevation in adduction of the left eye than of the right eye may be explained by the fact that the left eye was slightly amblyopic as compared with the right eye, and thus more

dissociated. The greater vertical deviation is usually found in the more dissociated eye. After surgical correction the eyes were straight for distance and near, with normal difference in the pupillary size, demonstrating dissociation of accommodation and convergence, which was effected by the good amplitude of fusion.

*Comment.*—In the three cases described, bilateral recession of the medial rectus corrected the greater esotropia for near and downward gaze without causing exotropia for distance and upward gaze. These results indicate that such surgical

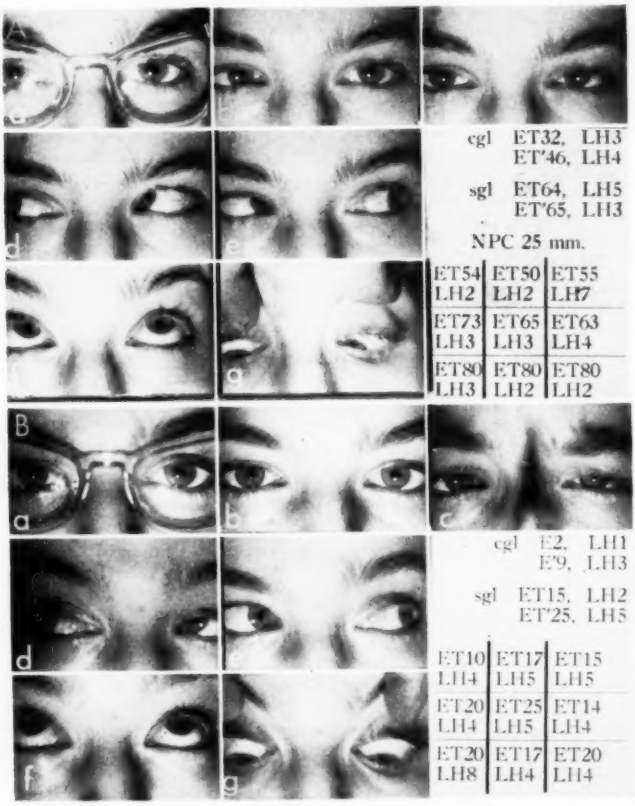

cgl ET32, LH3
ET'46, LH4
sgl ET64, LH5
ET'65, LH3
NPC 25 mm.
ET54 LH2 | ET50 LH2 | ET55 LH7
ET73 LH3 | ET65 LH3 | ET63 LH4
ET80 LH3 | ET80 LH2 | ET80 LH2
B
cgl E2, LH1
E'9, LH3
sgl ET15, LH2
ET'25, LH5
ET10 LH4 | ET17 LH5 | ET15 LH5
ET20 LH4 | ET25 LH5 | ET14 LH4
ET20 LH8 | ET17 LH4 | ET20 LH4

Fig. 3 (Case 3).—*A*, preoperative examination: (*a*) eyes straight for distance with glasses (demonstrated only when photographs were taken; eyes never observed to be straight in the clinic; pupils dilated, showing the patient was not accommodating); (*b*) 20 degrees of right esotropia for near without glasses; (*c*) 5 degrees left esotropia for near without glasses, with pupils more dilated than in *B*; (*d*) marked elevation of left eye in adduction; (*e*) elevation of right eye in adduction, which could be seen clinically but was not demonstrated well with photographs; (*f*) 15 degrees right esotropia looking up; (*g*) 25 degrees left esotropia looking down.

*B*, five months after bilateral 5-mm. recession of medial recti: (*a*) eyes straight for near with glasses, pupils small: (illumination same as in *A*); (*b*) eyes straight for near without glasses; small pupils; (*c*) convergence near point, 50 mm.; (*d*) elevation in adduction improved on gaze to the right; (*e*) elevation in adduction gone on gaze to left; (*f*) eyes straight, looking up; (*g*) eyes straight, looking down.

correction has its greatest effect on the near and downward positions of gaze. Since the recession of the medial recti alone corrected these squints in all positions of gaze, the underlying dysfunction must have been an overaction of the medial recti.[2] This may be due to convergence excess (Duane[3] and White[4]), paresis (Chavasse[5]), abnormal check ligaments (Scobee[6]) or congenital structural anomalies (Brown[7]).

Although the eyes may become straight in all positions immediately after recession of the medial recti, often the excess innervational impulses persist and the eyes are straight for distance and upward gaze immediately after operation, but some esotropia remains for near and downward gaze. One should not be hasty in reoperating for the remaining esotropia. Since the eyes are straight for distance and upward gaze, fusion may develop in these positions. As the fusional amplitude develops, excess innervational impulses may be controlled, and in time the eyes may become straight in all positions (Cases 1 and 2).

The elevation in adduction is either corrected or improved when the eyes are straightened horizontally by medial-rectus recessions only. This is evidence that the vertical deviations are probably secondary to the esotropia.

### BILATERAL RESECTION OF THE LATERAL RECTUS

CASE 4.—M. B., aged 8 years, came to the Infirmary on Nov. 10, 1947, with the history of esotropia since birth.

With atropine cycloplegia, refraction was as follows:

O. D.: + 1.00 sph. = 20/20
O. S.: + 1.50 sph. = 20/20

The results of examination are shown in Figure 4*A*.

On May 12, 1948, a bilateral 8-mm. resection of the lateral rectus was done.

Postoperative examination (June 4, 1948) showed 5 degrees of exotropia for distance and 15 degrees of esotropia for near.

Postoperative screening (Aug. 4) showed 15 △ of esotropia for distance and 25 △ of esotropia and 2 △ of left hypertropia for near.

| | | |
|---|---|---|
| XT4, LH5 | XT4, LH4 | XT5, RH2 |
| ET27, LH10 | ET25, LH12 | ET20, LH10 |
| ET28, LH4 | ET25, LH10 | ET25, LH10 |

On the synoptophore, fusion was second grade with amplitude.

The convergence near point was 95 mm.

The results of postoperative examination (Sept. 26, 1950) are shown in Figure 4*B*.

This case could be classified in Group I, even though preoperative screening measurements showed a left hypertropia only, and that in the wrong position for the group, namely, up and to the left. The diagnostic position is up and to the right. The other findings, such as the bilateral elevation in adduction and the greater amount of esotropia for near and down than for distance and up, placed the case in this group. Immediately after the bilateral resection of the lateral rectus muscle the eyes were divergent for distance and upward gaze, while an esotropia remained for near and downward gaze. Two years after surgical correction, the overaction of the medial recti was still present, as evidenced by the esotropia for near and downward gaze. The eyes

3. Duane, A.: Fuchs's Textbook of Ophthalmology, Ed. 7, Philadelphia, 1923, J. B. Lippincott Co.

4. White, J. W.: What Is the Minimum Routine Examination of Muscles, Arch. Ophth. **24**:1110 (Dec.) 1940.

5. Chavasse, B.: Worth's Squint, Ed. 7, P. Blakiston's Son & Co., London, 1939.

6. Scobee, R. G.: The Fascia of the Orbit, Am. J. Ophth. **31**:1539, 1948.

7. Brown, H. W.: Congenital Structural Anomalies, in Strabismus Ophthalmic Symposium, edited by James A. Allen, St. Louis, C. V. Mosby Company, 1950, p. 205.

could be straight for distance, with slight exotropia on looking up. Only a 10-degree improvement in the esotropia was present on looking down. Note that immediately after operation screening measurement showed bilateral hypertropia—left hypertropia to the right and right hypertropia to the left.

The results obtained may be due to surgically produced overaction of the lateral recti acting against primary overaction of the medial recti. It is quite possible that the horizontal position the eyes assume in different positions of gaze might depend on the interplay of the medial and the lateral recti and the dominance of one or the other group in these positions. If a medial-rectus recession were to be done for the remaining esotropia for near, the interplay of the

Fig. 4 (Case 4).—*A* (preoperative examination): (*a*) 10 degrees of right esotropia for distance; (*b*) 20 degrees of right esotropia for near; (*c*) convergence near point, 30 mm.; (*d*) and (*e*) slight elevation in adduction, seen clinically but not shown well with photographs; (*f*) eyes straight looking up (patient can have 5 to 10 degrees of esotropia looking up); (*g*) 35 degrees of left esotropia looking down.

*B* (Sept. 26, 1950), two years after bilateral 8-mm. resection of lateral recti, on May 12, 1948: (*a*) 15 degrees of right esotropia for near; (*b*) eyes straight for distance; (*c*) convergence near point, 25 mm.; (*d*) slight elevation of left eye in adduction; (*e*) eyes level on gaze to left; (*f*) 5 degrees of divergence of right eye looking up; (*g*) 25 degrees left esotropia looking down.

muscle groups would be changed, so that the overaction of the lateral recti, would become more dominant, and an exotropia could develop, especially for distance and upward gaze. An example of this result of the recession following the bilateral resection will be presented and discussed in the report of Case 5.

CASE 5.—J. L., aged 10 years, came to the Infirmary on Sept. 1, 1947, with a history of crossed eyes since the age of 18 months.

With atropine cycloplegia, refraction was as follows:

O. D.: + 3.00 sph. = 20/20
O. S.: + 3.00 sph. = 20/20

The results of examination are shown in Figure 5.

On Oct. 1, 1947, a bilateral 10-mm. resection of the lateral rectus was done.

The results of postoperative examination, two months after operation, are shown in Figure 6*A*.

On Jan. 27, 1948, a 5-mm. recession of the left medial rectus was done.

The results of postoperative examination (July 20, 1950) are shown in Figure 6*B*.

The bilateral resection of the lateral rectus straightened the eyes for distance and upward gaze (Fig. 6*A*, *a*, *g*). The 5-mm. recession of one medial rectus, done to correct the remaining esotropia for near, resulted in a pronounced exotropia for distance and upward gaze (Fig. 6*B*, *a*, *h*). In the intermediate position, that is, the position somewhere between 20 ft. and 13 in. (6 meters and 33 cm.), with the eyes looking straight ahead, the eyes could be straight. After the development of the exotropia, vertical components could be screened which were not apparent before the operation. The vertical deviations then appeared to be secondary to the exotropia. The post operative exotropia might be explained as follows:

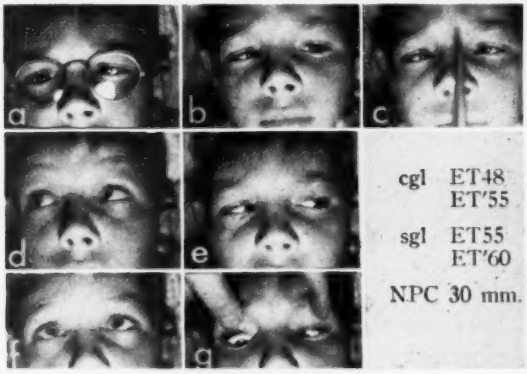



Fig. 5 (Case 5; preoperative examination).—(*a*) 35 degrees of left esotropia for near with glasses; (*b*) 35 degrees of right esotropia for near without glasses; (*c*) convergence near point, 30 mm.; (*d*) and (*e*) slight elevation in adduction seen clinically, not shown well with photographs; (*f*) 25 degrees of esotropia looking up; (*g*) 45 degrees of esotropia looking down.

After the bilateral resection of the lateral rectus, overaction of the lateral recti was produced. The interplay between the primary overaction of the medial recti and the surgically induced overaction of the lateral recti resulted in the eyes becoming straight for distance and upward gaze (lateral recti predominant) and remaining convergent for near and downward gaze (medial recti predominant).

After the 5-mm. recession of the left medial rectus to correct the esotropia for near, the interplay between the muscles was changed. The eyes became divergent for distance and upward gaze. It is not unlikely that the exotropia may have been caused by the release of some of the overaction of the medial recti, allowing the overaction of the lateral recti to become more pronounced. It was not due to an overrecession of a medial rectus with weakening of convergence, for the following reasons: (1) the good near point of convergence remaining; (2) the remaining esotropia for near and downward gaze, demonstrating that all the overaction of the medial recti had not been relieved with the 5-mm. recession; (3) the exotropia with bilateral elevation in adduction. Cases in this group have the greatest exotropia for distance and upward gaze. The cause of the exotropia is usually overaction of the lateral recti (divergence excess).

Fig. 6 (Case 5).—*A* (Oct. 1, 1947), two months after a bilateral 10-mm. resection of lateral recti: (*a*) eyes straight for distance with glasses; (*b*) 15 degrees of left esotropia for distance without glasses; (*c*) 25 degrees of left esotropia for near; (*d*) slight elevation of left eye in adduction; (*e*) slight elevation of right eye in adduction; (*f*) convergence near point, 35 mm.; (*g*) eyes straight looking up; (*h*) 25 degrees of right esotropia looking down.

*B* (Jan. 27, 1948), two years after reoperation with a 5-mm. recession of left medial rectus: (*a*) 10 degrees of left exotropia for distance; (*b*) eyes straight in intermediate position without glasses; (*c*) 15 degrees of right esotropia for near; (*d*) elevation of left eye in adduction; (*e*) slight elevation of right eye in adduction; (*f*) convergence near point 50 mm.; (*g*) 5 degrees of esotropia looking down; (*h*) 10 degrees of exotropia looking up; (*i*) 10 degrees of right esotropia for near.

In the intermediate position, where an exact balance between the overacting lateral recti and the overacting medial recti can obtain, the eyes can be straight.

CASE 6.—S. B., aged 3½ years, came to the Infirmary in 1940, with a history of gradual development of crossing of the left eye since the age of 1 year.

With atropine cycloplegia, refraction was as follows:

O. D.: + 1.25 sph. ⊂ + 1.00 cyl., ax 105 = 20/30
O. S.: + 1.50 sph. ⊂ + 1.00 cyl., ax 85 = 20/50

With occlusion of the right eye vision in the left eye improved to 20/30.

Examination showed that the eyes were close to straight for distance and had 15 degrees of esotropia for near. The patient could hold the eyes straight for distance and for near at times.

Screening measurements, with glasses, when vision was 20/30 in each eye, with the right eye dominant, revealed 17 △ of esotropia and 4 △ of left hypertropia for distance and 25 △ of esotropia and 4 △ of left hypertropia for near. Without glasses, when vision was unequal in the two eyes, being 20/40 in the right eye and 20/30 in the left eye, with the left eye dominant after occlusion of the right eye, there were 13 △ of esotropia and 3 △ of right hypertropia for distance and 22 △ of esotropia and 3 △ of right hypertropia for near.

Fusion was third grade at the objective angle; there was no hyperphoria; retinal correspondence was normal with the Tschermak cross.

Orthoptic training was given for two years.

Screening measurements (July 17, 1944) showed, without glasses, 22 △ of esotropia and 6 △ of left hypertropia for distance and 30 △ of esotropia and 3 △ of left hypertropia for near and with glasses 15 △ of esotropia and 3 △ of left hypertropia for distance and 21 △ of esotropia and 3 △ of left hypertropia for near.

| | | |
|---|---|---|
| ET20, LH6 | ..... | ET14, LH4 |
| ET27, LH1 | ET31, LH3 | ET27 |
| ET27 | ..... | ET27 |

On Aug. 10, 1944, a bilateral 5-mm. resection of the lateral rectus was done.

Postoperative examination (Aug. 12) showed 10 degrees of exotropia for distance and 5 degrees of esotropia for near.

Screening tests (Aug. 22) showed 16 △ of exotropia and 2 △ of left hypertropia for distance and 5 △ of esotropia and 1 △ of left hypertropia for near.

Postoperative orthoptic training was given for two years.

Examination (July 29, 1946) showed no deviation for distance or near, with or without glasses. Fusion was third grade at zero, with amplitudes to 24 △ base-out.

The child was discharged as cured.

On May 8, 1950, six years after operation, the status was as seen in Figure 7*A*.

With atropine cycloplegia, refraction was as follows:

O. D.: — 1.00 sph. ⊂ + 0.50 cyl., ax 60 = 20/40
O. S.: — 0.25 sph. ⊂ + 0.37 cyl., ax 90 = 20/15

The delayed cover test showed slight alternating hypophoria.

On July 27, 1950, a bilateral 7-mm. recession of the lateral rectus was done.

The postoperative results three weeks later are seen in Figure 7*B*, and those six months later, in Figure 7*C*.

In this case of esotropia with bilateral elevation in adduction, there was a definite convergence excess; the eyes could be straight at times and have a pronounced esotropia at other times. When the vision was reduced in the right eye, screen tests showed a right hypertropia. When vision became equal after occlusion of the left eye, screening showed a left hypertropia. She had two years of orthoptic training but still had a marked esotropia most of the time. A bilateral 5-mm. resection of the lateral recti was done for the intermittent esotropia. As the eyes could be straight at times, the recession of the medial recti was not considered because of the fear of postoperative divergence. Immediately after operation the eyes became divergent for distance. An esotropia remained for near. After two years of orthoptic training, screening showed orthophoria for distance and near, and she had third-grade fusion. The postoperative result may be explained as follows:

The bilateral resection caused an overaction of the lateral recti, which was manifested in exotropia for distance, since the eyes could be straight for distance before operation. The over-

Fig. 7 (Case 6).—*A*, examination six years after a bilateral 5-mm. resection of the lateral rectus muscle: (*a*) 25 degrees of right exotropia for distance with glasses; (*b*) 35 degrees of right exotropia for distance without glasses; (*c*) eyes straight for near; (*d*) and (*e*) slight bilateral elevation in adduction, seen clinically, but not shown well in photographs; (*f*) convergence near point 50 mm.; (*g*) 35 degrees of right exotropia looking up; (*h*) 5 degrees of right exotropia looking down; (*i*) 10 degrees esotropia looking down.

*B*, three weeks after bilateral 7-mm. recession of the lateral recti: (*a*) eyes straight for distance; (*b*) 20 degrees of right esotropia for near; (*c*) 5 degrees of right esotropia looking up (eyes can also be straight looking up); (*d*) 10 degrees of right esotropia looking down.

*C*, six months after surgical correction: (*a*) eyes straight for near with glasses; (*b*) eyes straight for distance without glasses; (*c*) convergence near point 50 mm.; (*d*) eyes level on gaze to the right, no limitation in abduction of right eye; (*e*) slight elevation of right eye in adduction, no limitation in abduction of left eye; (*f*) 5 degrees of right exotropia looking up; (*g*) eyes straight looking down.

action of the medial recti was not completely reduced, and an esotropia remained for near. It seems that the interplay of the overactions of the two groups could become balanced by means of orthoptic training and the development of good fusion amplitudes, so that two years after operation orthophoria was present for distance and near, with third-grade fusion. The patient may still have had an exotropia on upward gaze and an esotropia on downward gaze, as these positions were not studied at that time.

Six years after operation, her squint could be classified as exotropia with bilateral elevation in adduction caused by surgically produced overaction of the lateral recti. A slight esotropia could be seen at times looking down (Fig. 7*A*, *i*). This may mean that some overaction of the medial recti was still present. The interplay of the horizontal muscle groups changed so that the overaction of the lateral recti became more pronounced. This may have been due to lessening of the excess action of the medial recti with growth changes,[5] such as the change of the refraction from hypermetropia to myopia, the enlargement of the eyes, the enlargement of the face, and the development of a wide interpupillary distance.

A bilateral 7-mm. recession of the lateral rectus was done for the divergence excess of 30 degrees for distance. Immediately after surgical correction (Fig. 7*B*) there was an esotropia for near (*b*) and downward gaze (*d*). The eyes could be straight for distance (*a*) and looking up or could have a slight esotropia (*c*). The esotropia was probably not due to the overrecession of the lateral recti with weakening of divergence. It might be explained by a change in the interplay of the overacting medial and lateral recti. The overaction of the medial recti became more pronounced with recession of the lateral recti. Evidence for this follows:

1. The eyes were straight for distance and upward gaze.
2. There was no limitation of the lateral recti in abduction.
3. The overaction of the medial recti diminished as fusion developed from distance into the near positions of gaze with the straightening of the eyes (Fig. 7*C*, *a*).
4. As the eyes became straighter after operation, the esotropia became less on looking down (Fig. 7*B*, *d to C*, *g*). This lessening of the medial-rectus action resulted in change from slight estropia on looking up (7*B*, *c*) to slight exotropia looking up (7*C*, *f*). This shows that good function of the lateral recti was present.

*Comment.*—The effect of surgically induced overaction of the lateral recti for the correction of overaction of the medial recti has been demonstrated in Cases 4, 5, and 6. In summary, the surgically induced overaction of the lateral recti seems to have its greatest effect on the distance and upward positions of gaze. This was demonstrated by the eyes becoming either straight or divergent in those positions after operation, depending upon whether there was a large or a small preoperative esotropia in these positions. The unrelieved primary overaction of the medial recti was greatest in the near and downward positions of gaze, as shown by the small effect on the esotropia in these positions. In the intermediate position the eyes could be straight, divergent, or convergent, depending on the interplay of the overacting muscles and the dominance of either convergent or divergent impulses.

It is a good surgical principle to weaken overacting and to strengthen underacting muscles. The resection was contrary to this principle when used in cases of this group, since it produces overaction of the lateral recti in correcting a primary overaction of the medial recti. This operation has been used because of the reluctance of many surgeons to do the recession, owing to their fear of loss of convergence with ensuing exotropia. The resection was felt to be a safe procedure because the medial recti were not touched. However, in this group of cases, in which the esotropia for distance and upward gaze can be of very small amount, even a small bilateral resection can produce an exotropia (Case 6). The effect of the operation is of great interest because it permits us to study the position the eyes assume when two antagonistic groups of muscles are overacting at the same time.

UNILATERAL RECESSION OF THE MEDIAL RECTUS AND RESECTION OF A LATERAL RECTUS

CASE 7.—C. B., aged 4 years, came to the Infirmary on July 27, 1948, with the history of crossed eyes since the age of 18 months.

With atropine cycloplegia, refraction was as follows:

O. D.: $+3.00$ sph. $\bigcirc +0.75$ cyl., ax $90 = 20/30$
O. S.: $+3.00$ sph. $\bigcirc +0.50$ cyl., ax $90 = 20/30$

Examination showed 20 degrees of esotropia for distance and 25 degrees for near (Fig. 8*A*).

Fig. 8 (Case 7).—*A*, preoperative photographs: (*a*) 20 degrees of left esotropia for distance with glasses; (*b*) 25 degrees left esotropia for near without glasses; (*c*) convergence near point 30 mm.; (*d*) and (*e*) bilateral elevation in adduction.

*B* (July 17, 1950), one and one-half years after a 5-mm. recession of left medial rectus and an 8-mm. resection of left lateral rectus: (*a*) 20 degrees of right exotropia for distance with glasses; (*b*) eyes straight for near with glasses; (*c*) 15 degrees of right esotropia for near without glasses; (*d*) eyes level on gaze to right with slight decrease in adduction of left eye; (*e*) eyes level on gaze to left; (*f*) convergence near point 35 mm.; (*g*) 20 degrees of right exotropia looking up; (*h*) 10 degrees of right esotropia looking down; (*i*) eyes straight in intermediate position without glasses.

The delayed cover test showed bilateral hypertropia.

On Feb. 4, 1949, a 5-mm. recession of the right medial rectus and an 8-mm. resection of the right lateral rectus were performed.

The results of postoperative examination on July 17, 1950, are shown in Figure 8*B*.

After surgical correction, an exotropia was present for distance and upward gaze (Fig. 8*B*, *a*, *g*) and an esotropia for near and downward gaze (Fig. 8*B*, *c*, *h*), and the eyes could be straight in the intermediate position without glasses (Fig. 8*B*, *i*) and for near with glasses (Fig. 8*B*, *b*). A possible explanation of these changes may be that before operation the squint was small on looking up (esotropia of 27 △), and perhaps also for distance, even though the measurements were 49 △ of esotropia and 8-mm. resection overcorrected these positions. For near, the esotropia remained because the unilateral recession was insufficient to eliminate completely the overaction of the medial recti. In the intermediate position, the overaction of the medial and lateral recti could become balanced, and the eyes could be straight without glasses. A bilateral 4-mm. recession of the medial rectus would most likely have been the best operation in this case. It would have relieved the overaction of the medial recti without causing an exotropia for distance and upward gaze. The improvement of the right hypertropia after operation on the lateral muscles only is to be noted. It is also to be noted that before operation the delayed cover test showed a bilateral hypertropia but screening showed only right hypertropia. The right hypertropia was greatest in the diagnostic position and direction for the group, i. e., up and to the left.

Case 8.—M. K. P., aged 2½ years, came to the Infirmary on Nov. 29, 1940, with a history of esotropia since the age of 1 year.

With atropine cycloplegia, refraction was as follows:

O. D.: + 1.25 sph. ⊃ + 1.50 cyl., ax 45 = 20/200
O. S.: + 2.00 sph. ⊃ + 0.25 cyl., ax 85 = 20/25

With occlusion of the left eye, vision improved in the right eye to 20/25.

Perimetric measurements with glasses showed 23 degrees of esotropia for distance and 27 degrees for near; without glasses, 20 degrees for distance and 22 degrees for near.

Examination on July 19, 1943, showed 15 degrees of esotropia for distance and 25 degrees for near.

The convergence near point was 20 mm.

Screening measurements showed 30 △ of esotropia for distance and 45 △ of esotropia and 3 △ of right hypertropia for near.

| | | |
|---|---|---|
| ET55, LH3 | ..... | ET55, RH5 |
| ET50, LH7 | ET45, LH3 | ET55, RH3 |
| ET60, LH2 | ..... | ET60, RH3 |

Fusion was second grade.

On Sept. 22, 1943, a 4-mm. recession of the right medial rectus and an 8-mm. resection of the right lateral rectus were performed.

The results of postoperative examination, on May 25, 1948, are shown in Figure 9*A*.

On Aug. 7, 1948, a 5-mm. recession of the left medial rectus and an 8-mm. resection of the left lateral rectus were done.

Postoperative examination, on Oct. 18, 1948, showed exotropia of 20 △ for distance and 10 △ for near.

| | | |
|---|---|---|
| XT30, LH3 | XT36 | XT40, LH14 |
| XT25, LH2 | XT10 | XT24, LH6 |
| XT10, LH7 | 0 | LH3 |

The results of examination on June 26, 1950, are shown in Figure 9*B*.

The convergence near point was 110 mm.

The delayed cover test revealed alternating hypertropia.

The first operation reduced to a small amount the squint for distance and upward gaze (Fig. 9*A*, *b*, *g*). A large amount of esotropia remained for near and downward gaze (Fig. 9*A*, *c*, *h*). Since the overaction of the medial recti was not completely corrected by the 4-mm. recession, a 6-mm. recession of the left medial rectus was indicated. The resection of the left lateral rectus was contraindicated, since it could overcorrect the small squint for distance and upward gaze. A 5-mm. recession of the left medial rectus and an 8-mm. resection of the left lateral rectus were done. The result was as might be anticipated. Immediately after operation

there were a marked exotropia for distance and a small amount of exotropia for near. After two years of convergence exercises, an exotropia was still present for distance and upward gaze, although reduced in amount, and an exophoria was present for near. An esotropia could still be present on looking down (Fig. 9*B*, *f*). The divergence can be classified as exotropia with bilateral elevation in adduction. To correct the exotropia, a 7-mm. recession of one lateral rectus is advisable, since this type of exotropia is usually due to overaction of the lateral recti.

The predominance of the left hypertropia on screening was due to occlusion of the left eye for right amblyopia. When the vision in the eyes was about equal, screening showed bilateral hypertropia. This case demonstrates that a knowledge of the amount of squint for distance and

Fig. 9 (Case 8).—*A* (May 28, 1948), five years after a 4-mm. recession of right medial rectus and an 8-mm. resection of right lateral rectus (Sept. 22, 1943): (*a*) 25 degrees of right esotropia for near with glasses; (*b*) 15 degrees of right esotropia for distance with glasses; (*c*) 25 degrees of right esotropia for near without glasses; (*d*) convergence near point 30 mm.; (*e*) and (*f*) marked bilateral elevation in adduction; (*g*) 10 degrees of esotropia looking up; (*h*) 40 degrees of right esotropia looking down.

*B*, two years after operation 2, a 5-mm. recession of left medial rectus and an 8-mm. resection of left lateral rectus: (*a*) 10 degrees of right exotropia for distance (eyes can be straight for near); (*b*) convergence near point, 100 mm. with glasses; (*c*) 25 degrees of right exotropia looking up; (*d*) slight elevation of left eye in adduction with some limitation of left eye in adduction; (*e*) elevation of right eye more marked, with excess abduction of left eye; (*f*) 10 degrees of left esotropia looking down (eyes can be straight looking down).

upward gaze and for near and downward gaze is a factor in helping one to decide which muscles should be operated on for the best result.

CASE 9.—L. S., aged 7 years, came to the Infirmary in 1949, with a history of crossed eyes since the age of 3 years.

With atropine cycloplegia, refraction was as follows:

O. D.: + 3.00 sph. ⌒ + 0.25 cyl., ax 90 = 20/20
O. S.: + 3.00 sph. ⌒ + 0.50 cyl., ax 80 = 20/30

Fig. 10 (Case 9).—*A*, preoperative photographs: (*a*) 20 degrees of right esotropia for near with glasses; (*b*) 15 degrees of left esotropia for distance; (*c*) 25 degrees of right esotropia for near without glasses; (*d*) and (*e*) slight bilateral elevation in adduction; (*f*) 10 degrees of left esotropia looking up; (*g*) 45 degrees of right esotropia looking down.

*B* (May 25, 1950), after 5-mm. recession of left medial rectus and 8-mm. resection of left lateral rectus, on Jan. 7, 1950: (*a*) 25 degrees of left exotropia for near; (*b*) 30 degrees of right exotropia for distance; (*c*) 35 degrees of exotropia looking up; (*d*) limitation of left eye in adduction; (*e*) elevation of right eye in adduction, excess abduction of left eye; (*f*) eyes straight looking down.

*C* (Aug. 18, 1950), after reoperation, with 7-mm. recession of left lateral rectus, on June 7, 1950: (*a*) 10 degrees of right exotropia for distance; (*b*) 5 degrees of left exotropia for near (eyes can be straight for near); (*c*) 15 degrees of left exotropia looking up; (*d*) limitation of left eye in adduction; (*e*) elevation of right eye in adduction, excess abduction of left eye improved; (*f*) 20 degrees of right esotropia looking down.

The results of examination are shown in Figure 10*A*.

On Jan. 7, 1950, a 5-mm. recession of the left medial rectus and an 8-mm. resection of the left lateral rectus were performed.

The results of postoperative examination are shown in Figure 10*B*.

The convergence near point, absent on looking straight ahead, could be elicited in looking down, and was remote.

On June 7, 1950, a 7-mm. recession of the left lateral rectus was done.

Results of postoperative examination (Aug. 18, 1950) are shown in Figure 10*C*.

After the first operation the eyes became divergent for both distance and near. Exotropia was present on looking up, and the eyes could be straight in looking down. There was a pronounced limitation of the left medial rectus in adduction; no convergence near point could be elicited on looking straight ahead. With these findings, it would be reasonable to consider that the exotropia might be due to the recession or to postoperative slipping of the medial rectus, with loss of convergence. Certain evidence, however, seemed to indicate that the overcorrection was due, at least partially, to the resection of the lateral rectus:

1. The eyes before operation were close to straight on looking up and for distance (Fig. 10*A*, *f*, *b*). A resection of the lateral rectus could have overcorrected these positions (Cases 7 and 8).

2. The marked increase in the left eye in abduction could mean overaction of the lateral rectus (Fig. 10*B*, *e*). Poor relaxation of the overacting muscle could prevent the eye from moving in (Fig. 10*B*, *d*).

3. The greater exotropia for distance and upward gaze is found in cases of exotropia due to overaction of the lateral rectus.

4. If the medial rectus had slipped, an exotropia would probably have been present on looking down; in this case the eyes were straight on looking down.

On the basis of the above evidence, a 7-mm. recession of the left lateral rectus was done. Postoperatively (Fig. 10*C*) there were 10 degrees exotropia for distance (*a*) and 15 degrees on looking up (*c*). The eyes could be straight for distance and near (*b*) with small amount of esophoria, 9 △ for distance and 3 △ for near. The convergence near point improved to 6 inches (15 cm.). The esotropia in the straight-down position (Fig. 10*C*, *f*) may be explained as follows:

The 5-mm. recession of the left medial rectus and the 8-mm. resection of the left lateral rectus overcorrected the left eye, so that an exotropia developed in all positions of gaze except straight down. The uncorrected overaction of the right medial rectus was sufficient to keep the eyes straight on looking down. After the second operation, on the left lateral rectus, the uncorrected overaction of the right medial rectus became apparent, and 20 degrees of esotropia developed on looking down.

The 7-mm. recession apparently did not correct the effect of an 8-mm. resection of the same muscle, as shown by (1) the exotropia at times on looking up and for distance, and (2) the remaining limitation of the left eye in adduction. This limitation was greater than that which ordinarily follows 5-mm. recession of a medial rectus. It would have been better to have done a bilateral 4-mm. recession of the medial rectus in the first place. This would have corrected the large esotropia for near and downward gaze without causing the exotropia for distance and upward gaze. It would seem that resection of a lateral rectus along with recession of a medial rectus is not a good procedure in a case of this type.

Recent examination, on Aug. 29, 1951, showed that the improvement has been continuous and that the recession of the lateral rectus was a good procedure. Cover-test measurements were 5 △ of esophoria and 1 △ of left hypertropia for distance and 6 △ of esophoria for near.

| | | |
|---|---|---|
| XT38, LH10 | XT20 | XT2, RH4 |
| XT20, LH4 | E6 | ET14 |
| ET5, LH2 | ET12 | ET24 |

Second-grade fusion was found on the troposcope with the objective and subjective angles at zero. The near point of convergence was 80 mm. The patient fused the Worth four-dot test. The eyes were straight for distance and near. There were 15 degrees of exotropia on looking up and 15 degrees of esotropia on looking down.

CASE 10.—R. K., aged 5 years, came to the Infirmary in 1949, with a history of development of crossed eyes following a fever at the age of 2½ years.

With atropine cycloplegia, refraction was as follows:

O. D.: + 4.50 sph. = 20/70
O. S.: + 4.50 sph. = 20/20

Occlusion of the left eye brought vision in the right eye up to 20/20.

The results of examination are shown in Figure 11*A*.

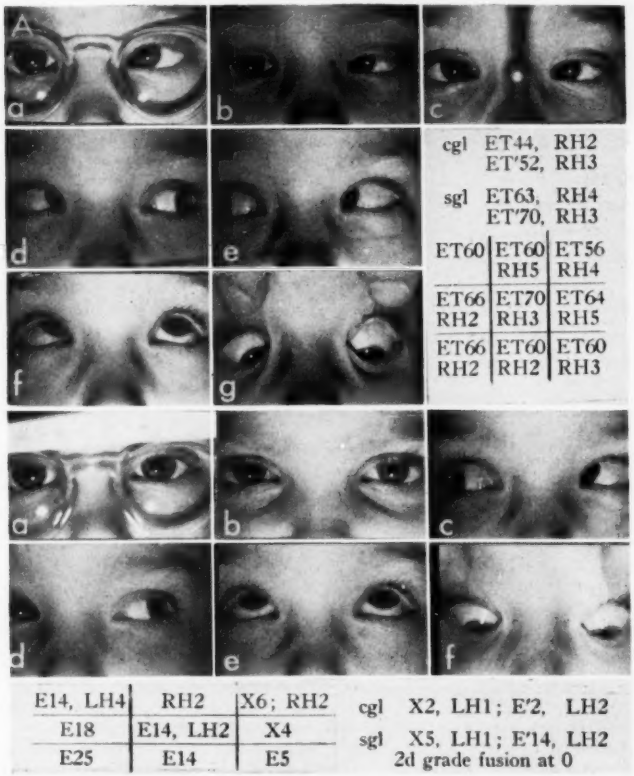



Fig. 11 (Case 10).—*A*, preoperative examination: (*a*) 20 degrees of left esotropia for near with glasses; (*b*) 25 degrees left esotropia for near without glasses; (*c*) convergence near point 25 mm.; (*d*), (*e*) bilateral elevation in adduction; (*f*) 25 degrees of right esotropia looking up; (*g*) 25 degrees of right esotropia looking down.

*B*, nine months after a 5-mm. recession of right medial rectus and an 8-mm. resection of right lateral rectus: (*a*) eyes straight for distance with glasses; (*b*) 5 degrees of right esotropia for near without glasses; (*c*) elevation of left eye in adduction; (*d*) elevation of right eye in adduction gone, limitation of right eye in adduction; (*e*) eyes straight looking up; (*f*) eyes straight looking down.

On Nov. 9, 1949, a 5-mm. recession of the right medial rectus and an 8-mm. resection of the right lateral rectus were done.

The results of postoperative examination are shown in Figure 11*B*.

This case can be classified as one of esotropia with bilateral elevation in adduction which has become concomitant, in that the esotropia is the same on looking straight up and on looking straight down. However, certain findings suggest, that the esotropia was noncomitant originally (Fig. 11*A*): (1) the esotropia of 66 △ down and to the right, as compared with the esotropia of 56 △ up and to the left, and (2) the slightly greater esotropia for near than for distance. The esotropia may have begun as a paretic or an accommodative squint (refraction, + 4.50 sph. for each eye) after a fever, at the age of 2½ years. Amblyopia developed in the right eye, tending toward its disuse. The right eye may have remained turned in most of the time, producing changes in the muscles and fascia. These structural changes of disuse tend to make a squint concomitant.[5]

A 5-mm. recession of the left medial rectus along with an 8-mm. resection of the left lateral rectus gave a good result. The resection helped the underaction of the lateral rectus, and the eyes became straight on looking up. The 5-mm. recession helped the overaction of the medial rectus, and the eyes became straight on looking down. When the squint is concomitant, especially when it is the same in looking straight up and in looking straight down, it may be best to work on the medial and the lateral rectus of one eye at the same time.

*Comment.*—When the squint is concomitant, especially when it is the same on looking straight up and on looking straight down, the recession-resection operation is a good procedure. For about 20 degrees of esotropia, a 5-mm. recession and an 8-mm. resection is correct. A study of the difference in the amount of squint for distance and upward gaze and that for near and downward gaze can be an important means of determining what type of surgery shall be done for reoperation. Operation on the lateral rectus can best affect the first position, while operation on the medial rectus can best affect the second position.

### RECESSION OF THE INFERIOR OBLIQUE COMBINED WITH OPERATION ON THE HORIZONTAL MUSCLES

CASE 11.—B. B., aged 4 years, came to the Infirmary in 1946, with the history of crossing of the right eye after measles, at the age of 1½ years.

With atropine cycloplegia, refraction was as follows:

O. D.: + 2.00 sph. ⊃ + 1.00 cyl., ax 105 = 20/50
O. S.: + 1.75 sph. = 20/30

The results of examination are shown in Figure 12*A*.

On July 12, 1947, a 4-mm. recession of the right medial rectus, an 8-mm. resection of the right lateral rectus, and an 8-mm. recession of the right inferior oblique were done.

The results of postoperative examination (March 22, 1948) are shown in Figure 12*B*, and those of later examination (Aug. 24, 1950), in Figure 12*C*.

This patient had bilateral elevation in adduction, as can be seen in Figure 12*A*, *d*, *e*, but screening showed only right hypertropia, most likely due to slight amblyopia in the right eye. Because of the finding only of the right hypertropia on screening, an 8-mm. recession of the right inferior oblique was done, along with a 4-mm. recession of the right medial rectus and an 8-mm. resection of the right lateral rectus. The 8-mm. resection straightened the esotropia for distance (2 △) and upward gaze (8 △); in fact, immediately after operation there was a tendency for the eyes to diverge on looking up and to the left (exotropia 12 △). The 4-mm. recession was insufficient to correct the esotropia for near and down. The 9-mm. recession of the inferior oblique produced paralysis of the right inferior oblique, as seen by the underaction of the right eye on looking up (Fig. 12*B*, *b*), and up and to the left (Fig. 12*B*, *c*). The left hypertropia became more pronounced on screening. Before operation the eyes were level looking up (Fig. 12*A*, *c*).

Examination three years after operation (Fig. 12*C*) showed that the esotropia had increased. This was most likely due to the persisting overaction of the left medial rectus. At present, (Aug., 1950) there are 15 degrees of esotropia for distance and 20 degrees of esotropia for near. The esotropia has increased on both looking up and looking down. The paresis of the right eye

Fig. 12 (Case 11).—*A*, preoperative photographs: (*a*) 30 degrees of right esotropia with right hypertropia for near; (*b*) 30 degrees of left esotropia with left hypertropia for near; (*c*) 20 degrees of right esotropia looking up (eyes on the same level); (*d*) and (*e*) bilateral elevation in adduction; (*f*) 40 degrees of left esotropia looking down.

*B*, nine months after 4-mm. recession of right medial rectus and 8-mm. resection of right lateral rectus, along with 9-mm. recession of right inferior oblique: (*a*) marked elevation of left eye up and to right; (*b*) slight esotropia looking up with marked lag of right eye; (*c*) lag of right eye up and to left; (*d*) 15 degrees of right esotropia for near; (*e*) 25 degrees of left estropia for near; (*f*) convergence near point 30 mm.; (*g*) marked elevation of left eye in adduction; (*h*) 25 degrees of right esotropia looking down; (*i*) limitation of the right eye in adduction; no elevation of the right eye in adduction.

*C*, three years after surgical intervention: (*a*) 20 degrees of right esotropia for near; (*b*) 15 degrees of right esotropia for distance; (*c*) convergence near point 30 mm.; (*d*) elevation of left eye in adduction; (*e*) elevation and limitation of right eye in adduction; (*f*) 5 degrees of right esotropia looking up with lag of right eye; (*g*) 35 degrees of left esotropia looking down.

is still present in looking up (*f*), and the elevation of the right eye in adduction has increased (*e*). Screening measurements now show the bilateral hypertropia to be greatest in the diagnostic directions of gaze for the group, i. e., left hypertropia to the right and right hypertropia to the left. This case demonstrates that screening in cases of bilateral elevation in adduction may show only one type of hypertropia at times and that it is a mistake to do a recession on one oblique because of this finding, for the following reasons: 1. The opposite hypertropia may become pronounced, and even greater than the original hypertropia. 2. A definite muscle paralysis is produced, adding a paretic primary hypertropia to hypertropias that were dissociated from, or secondary to, the horizontal deviation.

In this case a bilateral 4-mm. recession of the medial rectus only would most likely have given a better result. If the esotropia is corrected, the elevation in adduction may disappear. If, after a few years, the elevation in adduction remains and is cosmetically objectionable, a bilateral recession of the inferior oblique muscle would be of value as a secondary procedure.

Case 12.—W. B., aged 6 years, came to the Infirmary on Feb. 17, 1948, with the history that the right eye had turned in since December, 1946, after poliomyelitis.

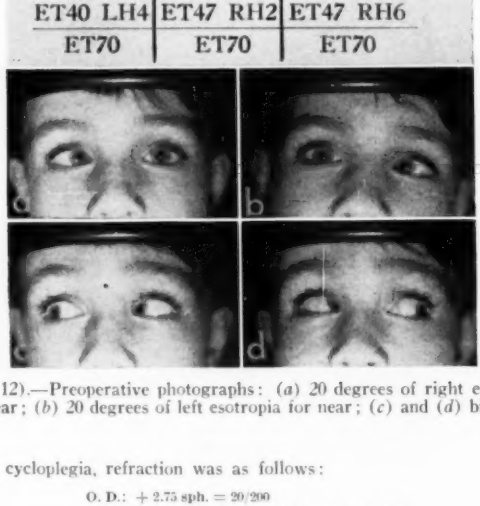



Fig. 13 (Case 12).—Preoperative photographs: (*a*) 20 degrees of right esotropia with right hypertropia for near; (*b*) 20 degrees of left esotropia for near; (*c*) and (*d*) bilateral elevation in adduction.

With atropine cycloplegia, refraction was as follows:

O. D.: + 2.75 sph. = 20/200
O. S.: + 2.25 sph. ⊃ + 0.50 cyl., ax 85 = 20/20

With occlusion of the left eye, vision in the right eye improved to 20/30.

Examination (Fig. 13) showed 15 degrees of esotropia for distance and 20 degrees for near.

In vertical versions, the eyes were nearly straight in looking up; with 35 degrees of esotropia in looking down.

The convergence near point was 30 mm.

On May 13, 1949, a 4-mm. recession of the right medial rectus, an 8-mm. resection of the right lateral rectus, and a 6-mm. recession of the right inferior oblique were performed.

The results of postoperative examination (Jan. 17, 1951) are shown in Figure 14*A*. Vision was 20/100 in the right eye and 20/20 in the left eye.

The convergence near point, absent when the eyes were looking straight ahead, was 40 mm. in looking down.

Occlusion of the left eye brought vision in the right eye to 20/20.

On March 20, 1951, a 7-mm. recession of the right lateral rectus was done.

The results of postoperative examination (June 20, 1951) are shown in Figure 14*B*.

This case shows that, although bilateral elevation in adduction was present, screening showed only right hypertropia in the primary position, probably because the right eye was amblyopic. The eyes were nearly straight on looking up and markedly convergent on looking down. A 4-mm. recession of the right medial rectus, an 8-mm. resection of the right lateral rectus, and a 6-mm. recession of the right inferior oblique were done, the last because of the dominance of the right hypertropia.

Fig. 14 (Case 12).—*A*, two years after 4-mm. recession of right medial rectus and 8-mm. resection of right lateral rectus, along with 6-mm. recession of right inferior oblique: (*a*) 15 degrees of left exotropia for distance and 15 degrees of exotropia for near (eyes at times can be close to straight for near); (*b*) convergence near point 40-mm. looking down (no convergence near point looking straight ahead); (*c*) 25 degrees of right exotropia looking up (lag of right eye looking up); (*d*) elevation of left eye in adduction; (*e*) slight elevation with limitation of right eye in adduction; (*f*) 5 degrees of left esotropia looking down.

*B*, examination three months after second operation, consisting of a 7-mm. recession of right lateral rectus: (*a*) 10 degrees of right exotropia for distance; (*b*) 5 degrees of right exotropia for near (eyes can be straight for near); (*c*) convergence near point 6 in. (15 cm.) looking straight ahead, 40-mm. on looking down; (*d*) elevation of left eye in adduction; (*e*) elevation and limitation of right eye in adduction; (*f*) 25 degrees of right exotropia with lag of right eye looking up (eyes can have 15 degrees of exotropia at times looking up); (*g*) 5 degrees of left esotropia looking down.

The results of the surgical procedure (Fig. 14*A*) can be explained as follows: The 8-mm. resection of the lateral rectus produced an exotropia for distance and upward gaze because the eyes were close to straight in these positions before operation. The 4-mm. recession almost eliminated the overaction of the medial recti on looking down. The unilateral recession of the inferior oblique caused the opposite hypertropia to become predominant. The left hypertropia increased from 2 △ up and to the right, to 26 △. It also produced a paralysis of the right eye on looking up (*c*). An interesting finding here is the absence of a convergence near point on looking straight ahead with a good convergence near point on looking down (*b*). This finding may indicate that the medial recti work best for near and downward positions of gaze.

Reoperation on the right lateral rectus has had its greatest effect on the straight-up position of gaze (Fig. 14*B*). It has reduced the exotropia from 54 to 32 △. The effect on the squint in looking down has been negligible, changing the esotropia from 5 to 8 △. It has had a marked effect also on the vertical deviations. The 26 △ of left hypertropia up and to the right has been reduced to 16 △ of left hypertropia. The right hypertropia has been increased from zero up and to the left to 10 △. There has been an improvement in the exotropia for distance, and the eyes can be straight for near a great deal of the time, with second-grade fusion. It seems, however, that the 7-mm. recession does not completely correct the effect apparently produced by the 8-mm. resection of the same lateral rectus.

CASE 13.—P. L. R., aged 8 years, came to the Infirmary on Jan. 19, 1950, with a history of poor vision in both eyes. She had been wearing glasses since the age of 2 years.

With atropine cycloplegia, refraction was as follows:

O. D.: + 6.00 sph. ⊃ + 2.00 cyl., ax 130 = 20/40
O. S.: + 7.00 sph. = 20/40

Screening measurements (Jan. 27, 1950), with glasses, showed 20 △ of esotropia and 14 △ of right hypertropia for distance and 30 △ of esotropia and 14 △ of right hypertropia for near, and, without glasses, 20 △ of esotropia and 14 △ of right hypertropia for distance and 30 △ of esotropia and 14 △ of right hypertropia for near.

| | | |
|---|---|---|
| ET18, RH16 | ET14 | ET18, RH10 |
| ET30, RH10 | ET30, RH14 | ET25, RH10 |
| ET30, RH8 | ET30 | ET25, RH6 |

Examination (Jan. 27, 1950) showed that the eyes could be straight with glasses and had 10 degrees of esotropia without glasses.

The results of examination on April 4, 1950 are shown in Figure 15.

Screening measurements showed, for the right eye, 25 △ of esotropia for distance and 30 △ for near and, for the left eye, 20 △ of esotropia and 25 △ of left hypertropia for distance and 30 △ of esotropia and 18 △ of left hypertropia for near.

| Right Eye: | | | Left Eye: | | |
|---|---|---|---|---|---|
| ET16 | ..... | ET18 | ET16 | ..... | ET18 |
| LH5 | ..... | ..... | LH30 | ..... | LH4 |
| ET18 | ..... | ET18 | ET18 | ET30 | ET18 |
| LH5 | ET30 | ..... | LH25 | LH18 | LH9 |
| ET18 | ..... | ET18 | ET18 | ..... | ET20 |
| ..... | ..... | ..... | LH16 | ..... | LH5 |

On May 8, 1950, an 8-mm. recession of the left inferior oblique and an 8-mm. resection of the left lateral rectus were done.

The results of postoperative examination on Aug. 9, 1950, are shown in Figure 16*A* and those on Feb. 28, 1951, in Figure 16*B*.

This case was one of intermittent accommodative squint with bilateral elevation in adduction. The eyes could be straight before operation (Fig. 15, *d*) at times. When screened, the eyes became dissociated, and large dissociated hypertropias could be measured—at times a right, and at times a left, hypertropia. Because of the large left-hypertropia screening measurements, a recession of the left inferior oblique was done, and an 8-mm. resection of the left lateral rectus was done for the small esotropia.

Postoperatively, the resection of the lateral rectus produced an exotropia for distance and near when she wore her correction (Fig. 16*A*, *b*). After she had been without her glasses for a while, an esotropia was present for distance and near (Fig. 16*B*, *a*, *b*). Whereas before operation, when she did not accommodate she could hold her eyes straight (Fig. 15*d*), now, when she does not accommodate she has a marked exotropia (Fig. 16*B*, *c*). The recession of the inferior oblique has produced a marked lag of the left eye on looking up. It has added a paretic component to what, before operation, were dissociated hypertropias.

This case demonstrates that a resection of a lateral rectus has its greatest effect on the screening measurements on upward gaze. In this case it changed the screening measurements from an esotropia of 18 to an exotropia of 15 △ on looking up and from an esotropia of 18 to an esotropia of 14 △ on looking down. In cases of this group, if the eyes are straight looking up, the resection of a single lateral rectus can cause an exotropia and is not a safe procedure for correction of a small esotropia.

CASE 14.—R. B., aged 14, came to the Infirmary in April, 1948, with a history of crossed eyes since the age of 2 years.

With atropine cycloplegia, refraction was as follows:

O. D.: + 1.00 sph. = 20/20
O. S.: + 1.00 sph. = 20/20

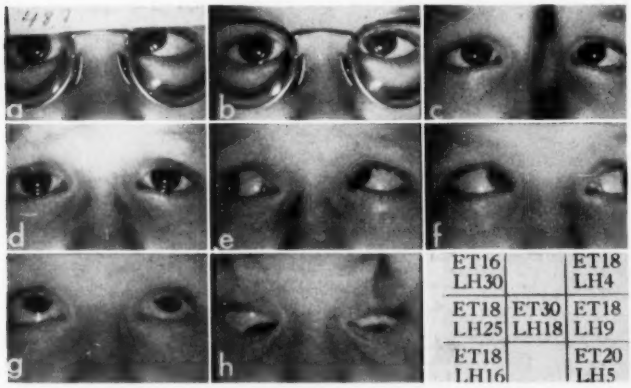



Fig. 15 (Case 13).—Preoperative examination: (*a*) 25 degrees of left esotropia (15 degrees of left hypertropia for near with glasses); (*b*) 10 degrees of left esotropia and 10 degrees left hypertropia for distance (eyes can be straight with glasses); (*c*) convergence near point 25-mm.; (*d*) eyes straight with glasses when not accommodating, no hypertropia; (*e*) and (*f*) bilateral elevation in adduction, more with left eye; (*g*) eyes straight looking up with slight elevation of left eye; (*h*) 15 degrees of right esotropia looking down.

The results of examination are shown in Figure 17*A*. Screening measurements showed 53 △ of esotropia and 14 △ of right hypertropia for distance and 55 △ of esotropia and 8 △ of right hypertropia for near.

On June 26, 1948, a bilateral 10-mm. resection of the lateral rectus muscle and a bilateral 10-mm. recession of the inferior oblique muscle were performed.

The postoperative results (July 20, 1948) are shown in Figure 17*B*. Screening tests showed 36 △ of esotropia and 5 △ of right hypertropia for distance and 52 △ of esotropia and 5 △ of right hypertropia for near.

On Dec. 16, 1948, a bilateral 4-mm. recession of the medial rectus was done.

The postoperative results (Jan. 10, 1951) are shown in Figure 17*C*.

The bilateral resection of the lateral rectus (Fig. 17*B*) had very little effect on the esotropia for near, reducing it only 3 △ on screening. It had a larger effect on the esotropia for distance,

reducing it 17 △. The bilateral recession of the inferior obliques reduced the right hypertropia 3 △ in the straight-ahead position and changed the elevation in adduction very little, as shown in Figure 17*B*, *d*, *e*.

After the bilateral 4-mm. recession of the medial rectus (Fig. 17*C*), the eyes became straight, the elevation in adduction disappeared in the left eye and became very slight in the right eye, and the patient had third-grade fusion with good amplitude. The disappearance of the elevation in adduction would seem to be due to the straightening of the eyes and the development of fusion following the operation on the medial rectus. There was enough esotropia for distance and up

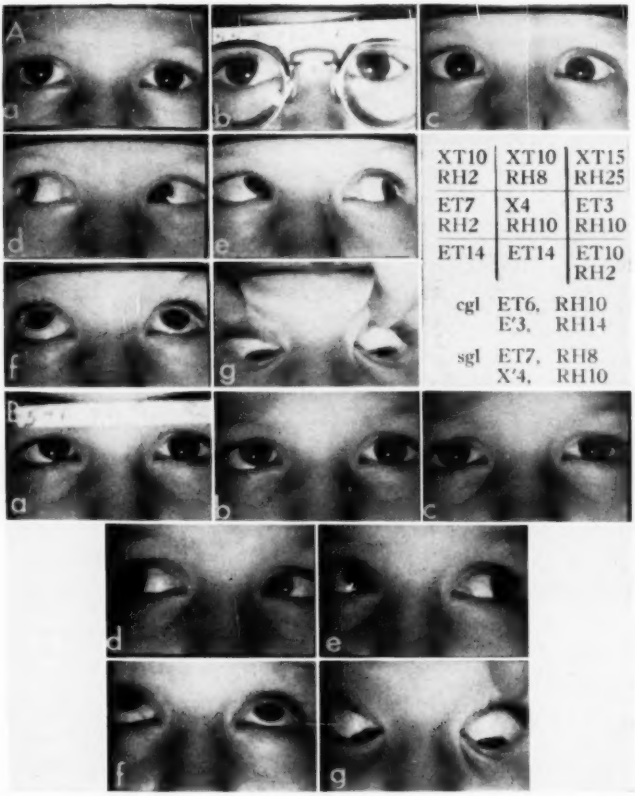



Fig. 16 (Case 13).—*A*, four months after 8-mm. recession of left inferior oblique and 8-mm. resection of left lateral rectus: (*a*) 10 degrees of exotropia and right hypertropia for near without glasses; (*b*) 20 degrees right exotropia and right hypertropia for distance with glasses; (*c*) convergence near point 50-mm.; (*d*) slight depression of left eye in adduction, relative right hypertropia in abduction; (*e*) marked elevation of right eye in adduction, excess action of left eye in abduction; (*f*) 20 degrees of right exotropia looking up with marked lag of left eye looking up; (*g*) 5 degrees of right esotropia looking down.

*B*, photographs taken after child had not worn glasses for one week, nine months after operation: (*a*) 15 degrees of right esotropia with right hypertropia for distance; (*b*) 15 degrees of right esotropia with right hypertropia for near; (*c*) 10 degrees of right exotropia for distance (not accommodating); (*d*) eyes level on gaze to right; (*e*) marked elevation of right eye in adduction; (*f*) 5 degrees of right exotropia looking up with marked lag of left eye; (*g*) 20 degrees of right esotropia looking down.

Fig. 17 (Case 14).—*A*, preoperative photographs: (*a*) 30 degrees of left esotropia with left hypertropia for near; (*b*) 30 degrees of right esotropia with right hypertropia for near; (*c*) convergence near point 30-mm.; (*d*) and (*e*) bilateral elevation in adduction, more on right; (*f*) 20 degrees of left esotropia looking up; (*g*) 40 degrees of right esotropia looking down.

*B*, one month after bilateral 10-mm. resection of lateral rectus muscles and bilateral recession of inferior oblique: (*a*) 25 degrees of left esotropia with left hypertropia for near; (*b*) 25 degrees of right esotropia with right hypertropia for near; (*c*) convergence near point 30-mm.; (*d*) and (*e*) bilateral elevation in adduction still prominent; (*f*) 20 degrees of left esotropia for distance.

*C*, two years after reoperation, which consisted of bilateral 4-mm. recession of medial recti: (*a*) eyes straight for near, stereopsis; (*b*) eyes straight for distance; (*c*) convergence near point 30-mm.; (*d*) elevation in adduction gone; (*e*) slight elevation in adduction; (*f*) eyes straight looking up; (*g*) eyes straight looking down.

before the first operation that the bilateral resection of the lateral rectus did not cause an overcorrection in these positions. There is a slight tendency toward an exotropia in looking up, as shown by the screening measurements on looking up and to the right (exotropia 8 △). The fusion amplitude has kept this tendency under control.

This case demonstrates that the horizontal squint may have to be corrected before the secondary vertical deviations disappear. The vertical-muscle surgery had little effect on the bilateral elevation in adduction. Only when the eyes became straight after the bilateral recession was the elevation in adduction reduced. The left-hypertropia measurements in positions up and to the right are still the same as before operation. It is possible that the operation on the medial recti only might have been sufficient to correct the elevation in adduction.

*Comment.*—The vertical components are dissociated hypertropias and are variable with screening. They are best elicited by screening each eye separately. The eye that is the more dissociated will usually show the greater screening measurement, and at times the only hypertropia. The more dissociated eye may be either the amblyopic eye or the eye that has been occluded. This seems to demonstrate the "Marlow effect" on dissociated eyes. The elevation in adduction will be more pronounced in the eye screening the predominant or only hypertropia. The bilaterality of the condition nearly always can be established by the demonstration of the opposite elevation in adduction. As demonstrated above, the unilateral recession of an oblique muscle because of the findings of only one type of hypertropia on screening causes the opposite hypertropia to become pronounced. It also causes a paresis of the recessed oblique muscle and adds a paretic component to dissociated hypertropias. A bilateral recession of the inferior oblique should be done if necessary. The attempted correction of the vertical deviation with the bilateral recession of the inferior oblique had very little effect on the esotropia, but the correction of the esotropia had a considerable effect on the reduction of the vertical components.

## CONCLUSIONS

1. In cases of esotropia with bilateral elevation in adduction and greater esotropia on looking down than up, there seems to be apparent overaction of the medial recti.

2. Bilateral recession of the medial rectus aimed at reducing the apparent overaction of these muscles gives the best surgical results.

3. Bilateral resection of the lateral rectus gives poor results in these cases, often causing an exotropia for distance with a residual esotropia for near. This is because one is not correcting the primary defect, but merely causing an overaction of the lateral recti, in addition to the overaction of the medial recti already present.

4. The recession-resection operation is a good procedure in cases of concomitant esotropia in which the squint is the same on looking up and on looking down. It is not a good procedure when the squint is noncomitant, in that there is a large esotropia on looking down and eyes are straight looking up. The resection of the lateral rectus tends to produce an exotropia in looking up and for distance, while the recession of the single medial rectus is insufficient to correct the large esotropia for near and downward gaze.

5. The recession of one inferior oblique during the primary operation does not give good results, for the following reasons: (*a*) The vertical deviations are

bilateral, and the recession of one oblique may cause a marked increase in the vertical deviation of the opposite type that was minimal and hard to detect before operation; (*b*) the vertical deviations may be secondary, and correction of the horizontal squint only may correct or improve them; (*c*) the recession may add a paretic vertical component to vertical deviations that were primarily dissociated or secondary to the horizontal squint.

6. Evidence has been presented which suggests that surgical procedures on the lateral recti have their greatest effect on the distance and upward positions of gaze, while operations on the medial recti have their greatest effect on the near and downward positions of gaze.

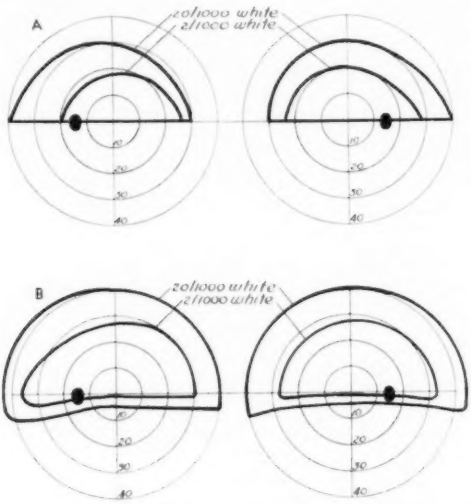
432½ Phoenix St.

# Registry of Interesting Cases

*It is the purpose of this section to record cases on the basis of interest only. No complete review of the literature is attempted and no claim for priority is implied except when specifically stated. Publication of case reports is subject to the concurrence of the Editorial Board on the interesting aspects of the case. It is requested that the reports submitted be as brief as is consistent with inclusion of the pertinent facts, bear a title that is as specific as possible, be prefaced by a paragraph that states why the author feels the case is of interest and be accompanied with photographs whenever possible.*

## ALTITUDINAL ANOPSIA FOLLOWING THORACOPLASTY

A case of bilateral inferior altitudinal anopsia is presented because of its infrequency and its sudden onset following a major operation. Anoxia of the superior lip of the calcarine fissure bilaterally is suggested as the probable cause.




Visual fields (*A*) in December, 1950 (vision 20/200— in each eye), and (*B*) July, 1951 (vision 20/200 + in each eye).

L. R. L., a business man aged 45, white, was referred by Dr. Seymour Farber because of poor vision following a second-stage thoracoplasty three and one-half months earlier.

There was a past history of syphilis in 1931, which was treated for one year with arsphenamine and "cured." He also gave a history of being on a starvation diet in a Japanese prison camp from 1942 to 1945.

Pulmonary tuberculosis was diagnosed in 1949, and, after a year in the Hassler Health Home, he was transferred to the San Francisco Hospital for surgical treatment.

On Aug. 16, 1950, a first-stage thoracoplasty was performed, with the use of nitrous oxide-ether anesthesia. The operation took four hours and was complicated by bleeding, which necessitated transfusion of 2 units of blood. The blood pressure had fallen from 170/90 to 90/60.

Several hours later there were severe dyspnea and unconsciousness for several minutes, which responded to suctioning and the use of an airway.

In spite of these complications during and after operation, recovery was prompt, and a second-stage thoracoplasty was performed Sept. 8. The latter operation took two and three-quarters hours. One thousand cubic centimeters of a dextrose solution and 1 unit of blood were administered during the operation, and the immediate postoperative condition was described as "fair." The blood pressure had fallen from 170/90 to 120/60.

The complaint of poor vision was first made some 20 hours after the second operation, and the patient was mildly disoriented for several days. Examination by the ophthalmology resident revealed vision to be limited to perception of hand movements in the right eye and to counting of fingers at 1 foot (30 cm.) in the left eye. Tension, fundi, and gross confrontation fields were normal. The neurological status, except for the ophthalmic findings, and the blood and spinal fluid were all within normal limits.

During the following two months vision slowly improved to a poor 20/200 in each eye.

Full therapeutic doses of streptomycin and paraaminosalicylic acid were given for three months, from Aug. 15 to Nov. 15, 1950.

On Dec. 26, 1950, the patient was first seen by us, at which time he exhibited bilateral inferior altitudinal anopsia (2/1000 and 20/1000 white). This defect was relative, as hand movements could be perceived in each lower field (Fig., *A*). There was, in addition, constriction of the 2/1000 white isopter to about 20 degrees for the upper field of each eye. The 2/1000 red test object could not be made out in any part of the field. The patient had never been color-blind. The fundi were normal except for faint sheathing of the retinal vessels near the disk. Manifest refraction and retinoscopy revealed a refractive error of —0.50 sph. in each eye. This correction did not improve the visual acuity of 20/200— in each eye. Neither eye was helped with a pinhole disk. Telescopic spectacles were of no help in reading. The Wernicke pupillary reaction to a slit-lamp beam was normal.

A neurological examination at this time showed nothing abnormal except for the visual loss The electroencephalogram was interpreted as normal.

Examinations every two months through July, 1951, have shown a very slight improvement in visual acuity to 20/200 + in each eye and in the visual fields (Fig., *B*).

The case was presented at the staff meeting of the Division of Ophthalmology, University of California School of Medicine in February, 1951, and the consensus was that the field defect was due to changes in the visual cortex which occurred during or just after the second thoracoplasty. There appeared to be no way of accurately evaluating the significance of the following possible contributory factors: (1) a history of syphilis, 1931; (2) starvation diet, 1942 to 1945; (3) nitrous oxide-ether anesthesia; (4) repeated blood transfusions; (5) anoxia incident to hemorrhage and pulmonary surgery; (6) three months of streptomycin-paraaminosalicylic acid therapy.

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# Annual Review

## CORNEA AND SCLERA

### Review of the Literature

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THIS REPORT consists of a review of the ophthalmic literature pertaining to the cornea and sclera which appeared in the latter part of 1950 and during the first eight months of 1951.

#### ANATOMY AND EMBRYOLOGY

As is to be expected, considerably less material has appeared on the anatomy and embryology of the cornea and sclera than on their clinical aspects.

Sebruyns [1] used the electronic microscope with magnifications of 15,000 to 20,000 diameters to show that living cells are much more complex than they were originally believed to be. Both nuclei and protoplasm contain innumerable capillary tubes and vacuoles, as well as granular matter. The capillary tubules and vacuoles are from 0.3 to 0.6 $\mu$ in diameter and vary in number and arrangement in different tissues. The basal membrane of the corneal epithelium has a spongy appearance, which is due to a vacuolar arrangement, resembling that in the renal glomeruli. The more anterior layers have a firmer structure. The stromal cells have a helical arrangement, which facilitates transparency.

Histologic preparations of the corneal endothelium were compared with biomicroscopic aspects by Busacca.[2] He showed that the endothelial mosaic is determined by cellular outlines, blurring of the mosaic is due to protoplasmic alterations, the endothelial cribrum is produced by a more or less pronounced hydropic degeneration of the cells, and the lymphocytes in the cellular deposits on the posterior wall of the cornea come from the aqueous humor and the histiocytes from the parenchyma.

Loewenstein [3] describes a "knob" at the edge of Descemet's membrane (lamina elastica posterior) and illustrates it with photomicrographs; this swelling is present between the margin of Descemet's membrane and the corneoscleral trabeculum. When aqueous enters the tissues, this area (the knob) may remain flat and form the white ring identical with the white ring of Schwalbe, or it may be swollen to form the white ring seen with the high-power slit lamp. It is granular and does not contain cells. The cornea from an eye injured by mustard gas (bis-[2-chloroethyl]sulfide) showed that the corneal lamellae were intensely swollen and dilated and a large knob, larger than in other cases, was present. This knob contained cloudy tissue, without any nuclei.

1. Sebruyns, M.: Ultrastructure of the Cornea and Lens Studied with the Electronic Microscope, Ann. ocul. **183**:483-494 (June) 1950.

2. Busacca, A.: The Corneal Endothelium in Biomicroscopy and in Histologic Preparations, London, Proceedings 16th International Congress of Ophthalmology, July, 1950, Vol. 2, pp. 767-772.

3. Loewenstein, A.: "Knob" at the Periphery of Descemet's Membrane, Brit. J. Ophth. **34**:246-250 (April) 1950.

PHYSIOLOGY, PHARMACOLOGY, AND EXPERIMENTAL PATHOLOGY

*Cornea.*—With the introduction of keratoplasty into ophthalmology and its growing therapeutic importance, it is not surprising that attention is being directed toward an understanding of the physiologic activities of the excised cornea and better means of storage. DeRoetth Jr.[4] has contributed work on the respiration and metabolism of the excised cornea. The metabolism of the cornea was studied under various storage conditions. It was observed that excised corneas maintained a normal carbohydrate breakdown for six days when they were stored in a moist chamber at 2 C. Under similar storage conditions, "intact" corneas or donor material in which the whole eye had been stored showed evidences of breakdown of protein after the first day of storage. This did not happen in the excised corneas until after the sixth day. There was also excessive hydration in the intact corneas, with resultant change in the corneal architecture. Oxygen uptake in both forms was normal for about six days. Glycolytic results were inconclusive, both for aerobic and for anaerobic activities. The conclusion, therefore, is that storage of the excised cornea is superior to storage of the intact cornea, although the excised state is inconvenient to handle for transportation purposes and it is also difficult to cut a disk from such a cornea with a trephine.

In an attempt to demonstrate that the excised cornea can utilize atmospheric oxygen, deRoetth used excised bovine, cat, and rabbit corneas and showed that they respired in air. In his experiments, the respiratory quotient of the bovine cornea in air was 1.00; the excised bovine cornea maintained a steady rate of respiratory activity in the Warburg flask regardless of the composition or pH of the suspending medium, the gas phase being either 100% oxygen or air. Homogenization of the corneal epithelium greatly decreased its ability to take up oxygen. The oxygen uptake of the homogenized corneal epithelium was increased by the addition of pyruvate and fumarate, but the rate of oxygen utilization was not affected by the addition of the same compounds.

Brief mention of the work of de Vincentiis and Auricchio[5] and that of Schaeffer and Murray[6] is indicated here; the former review briefly the recent studies on amino-acid metabolism and report their own investigations on the oxidation of amino acids in the ocular tissues. They measured colorimetrically the degree of deamination of the various acids and estimated the concentration of hydrazones after the addition of 2:4 dinitrophenylhydrazine. Among other parts of the eye, the cornea is able to deaminate several of the amino acids; the oxidation is carried on under the influence of specific oxidases. Schaeffer and Murray determined the amino-acid composition of the total proteins in the corneal stroma and epithelium and in the sclera of the bovine eye, first by isolating the individual proteins of these tissues and then by estimating the amino-acid composition of these proteins by the microbiologic assay method.

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4. deRoetth, A., Jr.: Metabolism of the Stored Cornea, Arch. Ophth. **44**:659-665 (Nov.) 1950; Respiration of the Cornea, ibid. **44**:666-676 (Nov.) 1950.

5. de Vincentiis, M., and Auricchio, G.: Metabolism of Amino Acids in Ocular Tissues, Acta ophth. **28**:7-22, 1950.

6. Schaeffer, A. J., and Murray, J. D.: Amino Acid Composition of Tissue Proteins of the Cornea, Sclera, Vitreous, and Retina, Arch. Ophth. **44**:833-841 (Dec.) 1950.

Experimental work on storage of the cornea bears a direct relation to that done on the metabolic activities of the stored cornea. Buschke,[7] alone and with other men, has contributed most to our knowledge of the best methods of storage; he tabulates in one paper the methods of corneal storage and the criteria of preservation. The work was done on fresh cows' eyes, and paraffin sections were made for histologic study from Zenker-fixed or formalin-alcohol-fixed material; control material was always used. The various storage methods used were as follows: (*a*) storage of the whole eye in a moist chamber over cotton soaked in isotonic sodium chloride solution; (*b*) storage of the whole eye in a moist chamber after replacement of the aqueous by liquid petrolatum U. S. P., N-hexadecane, or air; (*c*) storage of the excised cornea in closed vessels of 15- or 25-mm. capacity without added fluid; (*d*) storage of the excised cornea in liquid petrolatum, N-hexadecane, or N-octadecane; (*e*) storage of the cornea or of corneal disks of 10-mm. diameter in sealed 160-mm. vessels over 80 mm. of salt solutions of various concentrations. The storage temperature was between 2 and 7 C. throughout.

Histologic observations after storage showed the cell processes plainly and a balloon-like swelling and flattening of the nucleus, which had moved to one side of the cell; the cytoplasm showed earlier vacuolation. Storage of the whole eye in a moist chamber resulted in a significant uptake of water, noted eight hours after removal of the eye, with a great increase in the next few days; this uptake follows a regular, constant course. On the other hand, there was less fluid intake in whole eyes whose aqueous had been replaced. Isolated corneas suspended on a scleral lip took up much less fluid, but the minimal fluid uptake was found in corneal disks of 10-mm. diameter suspended over 0.5 mm. sodium chloride solution, after trial over various concentrations of aqueous solutions. In order to determine the state of preservation of corneal donor tissue stored under various conditions, Buschke, Howard, and Siegel[8] compiled quantitative data on nonprotein-nitrogen fractions in the tissue, which may eventually furnish useful criteria. Their report deals with data on total nonprotein nitrogen, ammonium nitrogen, and amino nitrogen in stored beef corneas.

Grignolo,[9] as well as Sezer,[10] tried cultivating corneal tissue, among other ocular tissues, on the chorioallantois of the chick embryo. Animal organisms react unfavorably to heterologous grafts, but corneal tissue had fairly good survival and showed proliferation of epithelium and stroma. The grafts were capable also of supporting the growth of the viruses of vaccinia and of herpes simplex.

Reviewed in the current literature is a book by Scuderi[11] which deals with corneal permeability. The author discusses the factors conditioning the permeability of the cornea and conjunctiva, the characteristics of absorption and the methods of research, the mode of absorption of drugs, the physiologic factors which

7. Buschke, W.: Studies on Experimental Corneal Storage, Am. J. Ophth. **34**:153-164 (May, Pt. 2) 1951.

8. Buschke, W.; Howard, A., and Siegel, L.: Nitrogen Fractions as Criterion of Preservation of Corneal Tissue, A. M. A. Arch. Ophth. **45**:27-31 (Jan.) 1951.

9. Grignolo, A.: Culture of Corneal, Conjunctival, Scleral, and Uveal Tissue in Chorioallantois of Chick Embryo, Boll. ocul. **29**:617-637 (Oct.) 1950.

10. Sezer, N.: Cultivation of Conjunctival and Corneal Tissue on Chorioallantoic Membrane, Arch. Ophth. **44**:703-709 (Nov.) 1950.

11. Scuderi, G.: Corneal Permeability, Edizione Minera medica, Torina, Italy, 1950.

influence permeability, the permeability of the various layers of the cornea, the permeability after administration of most of the organic and inorganic compounds employed in therapy, the chemical and chemicophysical factors exerting an influence on permeability, and external causes. The book is an exhaustive treatise on the subject, and very valuable to ophthalmology. At about the same time, Scuderi [12] published experimental work on the effect of ultraviolet radiation on corneal permeability. He concluded that there was greater permeability in the irradiated eyes as a result of exposure to the ultraviolet radiation and of disturbance of the Donnan equilibrium of the epithelium and the precorneal film.

Further experimental work was done for the determination of means of improving wound healing. Heerema and Friedenwald [13] found that wool fat U. S. P., which is an important component of ophthalmic ointment bases, produced a pronounced inhibition in the healing of minute wounds of the rat's corneal epithelium. A similar inhibition, but to a slighter degree, was produced by white petrolatum U. S. P. The authors found, however, that repeated washing of the wool fat with an appropriate aqueous washing fluid removes or destroys the toxic component without apparently seriously affecting its desirable physical characteristics.

Bonacorsi [14] conducted experiments to test the action of paraaminobenzoic acid on the repair of aseptic wounds of the cornea of rabbits. It has a decidedly favorable action both on the process of reepithelization and on the regenerative processes of the connective tissue. In an effort to improve regeneration of corneal epithelium, Marr, Wood, and Storck,[15] using the method of Buschke and Friedenwald, tried several agents on epithelial defects. The antibiotic derivatives of Actinomycetes did not delay healing of epithelial defects in the rat cornea; normal healing took place with crystalline penicillin G in a concentration of 2,500 u. per cubic centimeter, but a concentration of 50,000 u. per cubic centimeter was found to retard healing; bacitracin in a concentration of 1,000 u. per cubic centimeter also retarded healing. Healing was also prevented by two nonmetallic organic antiseptics, benzalkonium (zephiran®) chloride and benzethonium (phemerol®) chloride, and by two anesthetic solutions, butacaine sulfate U. S. P. and tetracaine hydrochloride U. S. P. The presence of ascorbic acid promoted healing.[16]

In a similar vein, several agents were used on corneal epithelium to determine their effect on intercellular cohesion [17]; chymotrypsin, trypsin, hyaluronic acid, ribosenucleic acid, higher alcohols, and long-chain nonionic polar-nonpolar substances were used. Since chymotrypsin and trypsin show such striking effects, the inference is drawn that proteins are important as substrates of the cohesive boundaries. Hyaluronic acid and ribosenucleic acid did not show evidence of participa-

12. Scuderi, G.: Effect of Ultraviolet Irradiation on Corneal Permeability, Boll. ocul. **29**:563-570 (Sept.) 1950.

13. Heerema, J. C., and Friedenwald, J. S.: Retardation of Wound Healing in the Corneal Epithelium by Lanolin, Am. J. Ophth. **33**:1421-1427 (Sept.) 1950.

14. Bonacorsi, A.: Effect of *p*-Aminobenzoic Acid on Repair of Experimental Lesions of the Cornea, Minerva chir. **5**:275-281 (May) 1950.

15. Marr, W. G.; Wood, R., and Storck, M.: Effect of Some Agents on Regeneration of Corneal Epithelium, Am. J. Ophth. **34**:609-612 (April) 1951.

16. Campbell, F. W., and Ferguson, I. I.: The Role of Ascorbic Acid in Corneal Vascularization, Brit. J. Ophth. **34**:329-334 (June) 1950.

17. Buschke, W.: Problems of Intercellular Cohesion in Corneal Epithelium, Proceedings 16th International Congress of Ophthalmology, July, 1950, Vol. 2, pp. 1363-1367.

tion as essential links in the mechanism of intercellular cohesion. The last two of the substances used also gave negative results as to participation of lipoid linkages in this mechanism. Buschke concluded that intrinsic proteolytic activity in the tissue accounted for the physiologic and pathologic changes in intercellular cohesion.

The prominence of corneal transplantation in ophthalmology has prompted the sudden influx of published experimental work. Rexed and Rexed [18] have reported their histologic studies on degeneration and regeneration of corneal nerves in the rabbit. Incisions varying from 90 to 360 degrees of the circumference of the cornea were made through the stroma down to Descemet's membrane, and then the sensitivity of the cornea was tested from the first day to six months. The eyes were then enucleated at varying intervals, stained with a silver preparation, and then studied. The authors found that the denervated insensitive area was always smaller than the full arc of the incision. The central cornea, however, did not become insensitive with incisions of less than 300 degrees of the circumference, which indicates extensive branching of the fibers. The regenerated nerves were slow to penetrate the scarred area, but after penetration did occur their progression was rapid. It was shown that the new fibers came from the severed nerves rather than from the adjacent, unsevered nerves. Regeneration began at the severed ends within three days, and the neurites generally invaded the stroma without regard to previous fiber paths; many entered and grew along old Schwann sheaths, but many remained naked within the stroma. This lack of directive growth by the sheaths of Schwann seems to constitute the greatest difference between the regeneration of corneal nerves and the regeneration of peripheral nerve fibers elsewhere in the body.

*Sclera.*—Again, experimental work on the sclera is minimal. The outstanding contribution is by Winkelman,[19] who reports the clinical and histologic findings in a rabbit in which a central round disk of the left cornea was replaced by a well-fitting disk of sclera from the right eye of the same animal. The graft took and assumed, to a large extent, the clinical characteristics of normal cornea. The clinical picture suggested that the scleral tissue of the transplant disappeared and was replaced by transparent or semitransparent corneal tissue. Histologically, the superficial layers of the graft were almost indistinguishable from normal cornea, while most of the deeper layers retained a structure characteristic of sclera. In a few places the scleral fibers were fragmented or necrotic. There was no decision as to whether the graft was being replaced by adjacent cornea or was being transformed into corneal tissue. This study may help to establish keratoplasty as a common procedure, since it is possible that if use of sclera becomes feasible the present problems met in obtaining donor material will be quickly and effortlessly solved.

## DIAGNOSIS AND THERAPY

Therapy has always been, and will always be, a most important part of the work of the clinical ophthalmologist. This is evidenced by the much larger volume of literature devoted to therapy during the current year than is found on other

18. Rexed, B., and Rexed, U.: Degeneration and Regeneration of Corneal Nerves, Brit. J. Ophth. **35**:38-49 (Jan.) 1951.

19. Winkelman, J. E.: Experimental Transplantation of Sclera, Ophthalmologica **119**:336-343 (June) 1950.

subjects. One of the most important contributions is on the use of corticotropin (ACTH) and cortisone in the treatment of various lesions affecting the cornea and sclera.

*Herpes of the Cornea.*—With the introduction of antibiotics into common therapeutic use, the treatment of herpes zoster ophthalmicus received new stimulation and impetus. Among the less-known methods of treatment are listed the use of ether and methenamine (hexamethylenetetramine). Kronenberg [20] recommends ether; his method is as follows: After local anesthesia and staining of the lesion with fluorescein, ether is rubbed on the ulcer with a cotton applicator. This treatment is repeated once or twice if spots of continued staining warrant it. Nitromersol (metaphen®) ointment and a pad and bandage are applied for at least 72 hours. The important points emphasized are rubbing, rather than merely applying, the ether, with repetition if necessary. He claims good results in the cases in which he used this therapy. Mehlhose and Oesterle [21] report that they successfully treated herpetic keratitis with daily intravenous injections of 10 cc. of methenamine in several cases. It is assumed that the formaldehyde which splits off from the methenamine destroys the causative virus. The recent literature on clinical manifestations and treatment of corneal herpes is reviewed by Offret[22]; he points out that a vast array of therapeutic methods is available, many of which he discusses in detail. (These include heat therapy, radiation therapy, application of chemical and antibiotic agents, subconjunctival injections, tarsorrhaphy, and lamellar keratoplasty.) Farnarier [23] also reviews some of the recent literature: He includes administration of cobra venom, use of vitamin B preparations, ionization, high-frequency and radiation therapy, regional injection of alcohol, and even resection of the greater superficial petrosal nerve. He believes that the greatest hope of abating or preventing the symptoms of the disease lies in the use of aureomycin or chloramphenicol. To date, the most successful therapeutic agents seem to be the new antibiotics; reports of good results are beginning to appear already.[24] Both local and parenteral administration were used; aureomycin was given in a 1% solution, two drops being instilled every two hours, and chloramphenicol, in a dose of 250 mg. every three or four hours around the clock. Rapid and marked improvement was noted in almost all cases. Chloramphenicol was administered orally; 0.75 gm. every four hours was given for two doses and then 0.50 gm. every four hours for four days. On the fourth day the dose was cut to 0.50 gm. every six hours, and administration was continued for 10 days. The symptoms of the disease were mitigated, and the course was considerably shortened.

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20. Kronenberg, B.: A Further Report on the Use of Ether in the Treatment of Herpetic Keratitis, New York J. Med. **50**:2825-2826 (Dec. 1) 1950.

21. Mehlhose, F., and Oesterle, G.: Hexamethylenetetramine in Herpetic Diseases of the Cornea, Klin. Monatsbl. Augenh. **117**:169-173, 1950.

22. Offret, G.: Treatment of Herpes of the Cornea, Année thérap. en ophtal. **1**:309-317, 1950.

23. Farnarier, G.: Treatment of Acute Sequelae of Ophthalmologic Zoster, Année therap. en ophtal. **1**:149-161, 1950.

24. Shier, J. M., and Provisor, B.: Epidemic Herpes Zoster Ophthalmicus, Am. J. Ophth. **33**:1921-1922 (Dec.) 1950. Pritikin, R. I., and Duchon, M. L.: Herpes Zoster Ophthalmicus: Report of Case Successfully Treated with Aureomycin, ibid. **34**:893-894 (June) 1951. Bruna, F.: Action of Aureomycin on Herpetic Infections of the Cornea, Boll. ocul. **29**:496-510 (Aug.) 1950. Beil, W. C.; Keith, F., and Mims, J. L., Jr.: Herpes Zoster Ophthalmicus Treated with Chloromycetin, Am. J. Ophth. **34**:623-625 (April) 1951

Herpes simplex is another disease of the cornea which, although apparently not so serious as herpes zoster ophthalmicus, can be somewhat debilitating and may result in serious sequelae. The emphasis at present seems to be on the use of aureomycin as the therapeutic agent of choice. Braley,[25] in his report, evaluates the use of aureomycin in recurrent herpes of the cornea and indicates it as the best method, instead of the previously used iodine cauterization. He found that the lesions were less serious and that healing usually occurred in 24 to 48 hours. His work was not substantiated by that of Geller and Thygeson,[26] who conducted experiments to test the effect of aureomycin, chloramphenicol, and terramycin in infections with the herpes simplex virus. They found that it was impossible to prevent or to modify experimental herpes keratitis in the rabbit with topical, intramuscular, or subconjunctival administration of aureomycin. Only intravenous administration seemed to modify it when the aureomycin was given in doses of 40 to 50 mg., combined with topical use. Subconjunctival administration of chloramphenicol was equally ineffective; large doses given orally may have been effective—in only two cases, however. Terramycin hydrochloride given orally also appeared to have no effect. The use of ergotamine tartrate and dihydroergotamine is advocated by Sĭmková,[27] who reports good results in 36 of 40 patients treated. Ergotamine tartrate was given internally in amounts varying from 3 to 6 mg. per day, and the dihydroergotamine (1 mg. in 4 cc. of sodium chloride) solution was placed on the cornea every two hours. Between applications, the eye was kept patched and atropinized.

Follicular hormone (estrone), given orally or implanted, was used in several cases of herpetic keratitis by Obal[28]; the spread of infections is inhibited by estrogens, a desirable effect in virus diseases and infections of the cornea.

*Ulcerative Keratitis.*—The treatment of ulcerative keratitis presents a difficult problem to the clinical ophthalmologist. Many times the available methods fail to halt the progress of the ulcer. Friede[29] believes that the treatment generally fails because the applied drug fails to reach the bacteria which lie in the progressively extending margin of the ulcer. The parenchyma is replete with fluid as a result of stasis, and this fluid exerts a strong hydrostatic pressure against the entrance of solutions of drugs. To dehydrate the tissue, the author applies a cotton applicator saturated with a strong solution of alcohol for two minutes to the cocainized ulcer; after 30 minutes, the ulcer is touched with an applicator dipped in an alcoholic solution of 5% azosulfamide ("prontosil"), 2% ethylhydroeupreine (optochin*), and 5% iodine. He repeats the procedure three to eight times a day, and progression of the ulcer is halted in 24 to 48 hours. Penicillin (2 minims [0.12 cc.] of 200,000 u. in 0.5 cc.) is injected into the cornea under the ulcer, infiltrating

25. Braley, A. E.: Evaluation of Treatment of Herpes Simplex Cornea with the Newer Antibiotics, J. Iowa M. Soc. **40**:486-490 (Oct.) 1950.

26. Geller, H. O., and Thygeson, P.: Aureomycin, Chloromycetin, and Terramycin in Experimental Herpes-Simplex Virus Infections, Am. J. Ophth. **34**:165-174 (May) 1951.

27. Sĭmková, M.: Treatment of Herpetic Eye Diseases with Gynergen and Dihydroergotamine, Ophthalmologica **119**:381-396 (June) 1950.

28. Obal, A.: Influence of Estrogens in the Eye, Arztl. Wchnschr. **5**:804-810 and 831-836, 1950.

29. Friede, R.: Medical and Operative Treatment of Serpiginous Ulcer, Klin. Monatsbl. Augenh. **117**:391-405, 1950.

the whole affected area. In chronic cases, the cornea is trephined around the ulcer, and the necrotized tissue down to Descemet's membrane is removed; dextrose powder is applied to the trephined area several times a day until regeneration occurs.

Since ascorbic acid is important to the repair of collagen tissues, its influence on the healing of corneal ulcers was studied by Boyd and Campbell.[30] They gave 1.5 gm. of ascorbic acid daily to one group and control tablets to another group of the same number. Ascorbic acid had no significant effect on the healing time of superficial ulcers but did accelerate the healing of deep ulcers. Apparently, there is a localized area around the site of the regenerating corneal collagen where the ascorbic-acid level falls below the optimum for rapid healing. Ascorbic acid, therefore, can be administered to promote healing.

Most authors have found, however, that the most uniformly good results are obtained when a combination of penicillin and sulfonamides is used. Penicillin is generally used locally and sulfonamides systemically. Bourlakova[31] advises using penicillin in pure cod liver oil up to 10,000 u. per milliliter. A combination of two sulfonamide drugs in ointment form (5% "supronalum" [a mixture of sulfamerazine and the 4-aminomethylbenzene sulfonamide salt of sulfathiourea]) was used by Straub,[32] with successful results.

Legrand[33] reviews the recent literature concerning treatment in the many cases of keratitis in which the response to the classic methods of treatment is not satisfactory. He discusses in detail the injection of penicillin and other substances into the anterior chamber; the use of conjunctival flaps, tarsorrhaphy, iridectomy, and keratectomy, as well as decompression, either by paracentesis or by a fistulizing operation, and tissue therapy. Even the use of the old-fashioned seton is reviewed; stress is put on the great value of corneal grafts for therapeutic purposes in conditions of this type.

*Epidemic Keratoconjunctivitis.*—Superficial punctate keratitis, or epidemic keratoconjunctivitis, is a distinct clinical entity involving both eyes. There are from 3 to 20 small, discrete epithelial opacities, usually in the pupillary area, and seen with the slit lamp. They stain poorly and are nonulcerative. Corneal sensitivity is reduced, but the symptoms are photophobia, burning, and blurring of vision. This disease is resistant to ordinary antiseptics, the sulfonamides, penicillin, streptomycin, tyrothricin, and aureomycin. Thygeson[34] is of the opinion that therapeutically valuable measures are (1) the application of 3.5% tincture of iodine after removal of the entire corneal epithelium, and (2) repeated vaccinations for smallpox used as in recurrent herpes simplex. All patients treated in this manner had alleviation of symptoms, and at least half showed complete healing.

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30. Boyd, T. A. S., and Campbell, F. W.: Influence of Ascorbic Acid on Healing of Corneal Ulcers in Man, Brit. M. J. **2**:1145-1148 (Nov.) 1950.

31. Bourlakova, T. N.: Penicillin in Treatment of Corneal Ulcers, Vestnik. oftal. **29**:11-13 (Nov.-Dec.) 1950.

32. Straub, W.: Treatment of Serpiginous Ulcer with "Supronalum" Ointment, Klin. Monatsbl. Augenh. **116**:187-191, 1950.

33. Legrand, J.: Surgical Treatment of Progressive Keratitis, Année thérap. en ophtal. **1**:223-256, 1950.

34. Thygeson, P.: Superficial Punctate Keratitis, J. A. M. A. **144**:1544-1549 (Dec. 30) 1950.

Hyaluronidase in an aqueous solution (5 turbidity-reducing units per cubic centimeter), was instilled into the conjunctival sac four to five times a day by Dekking [35] in cases of superficial punctate keratitis and was found to be very effective in limiting the disease.

*Corneal Vascularization.*—This subject does not properly fit in with discussion of diseases of the cornea, but since vascularization of the cornea so often follows many of the affecting disease processes, the treatment of this condition is important and deserves mention. In their last chapter on this subject, dealing with therapy, Offret and Chauvet [36] divide vascularization into superficial and deep types. Superficial vascularization is treated by (1) obliteration by cautery, (2) peeling, (3) retrobulbar injection of alcohol, and (4) phenolization of the sphenopalatine ganglion. Deep vascularization is treated by (1) galvanocautery and (2) death coagulation.

The complications of therapy are perforation into the anterior chamber and permanent damage to vision by gross hemorrhages into the corneal stroma. Where bunches of vessels occur, the method of choice is keratectomy, followed by total lamellar graft plus use of riboflavin and ascorbic acid.

*Ocular Tuberculosis.*—Tuberculosis of the eye may affect all parts of the eye and its adnexa; among the lesions found are sclerokeratitis and various keratitides of an ulcerative and deep nature. Streptomycin has proved to be of great value in treatment of the disease; it penetrates the ocular tissues readily whether it is used systemically or topically. In the cases reported by Parisi,[37] the patients received from 41 to 385 gm. of streptomycin intramuscularly over periods of from 39 to 187 days; the daily dose varied from 1 to 2 gm., given in divided doses. Thierry and Hevia [38] support his claim of good results. Woods, Wood, and Naquin [39] found that a combination of streptomycin and paraaminosalicylic acid is a striking deterrent to the course of experimental ocular tuberculosis in the rabbit.

*Other Forms of Therapy.*—A. Antibiotics and Chemotherapeutic Agents: It is advisable at this point to include work on the use of the newer drugs in ophthalmology. An excellent contribution has been made by Fried [40]; he has listed various useful drugs (sulfonamides, penicillin, streptomycin, bacitracin, and aureomycin) and has compiled a table of indications and methods of therapy for each. Penicillin is effective chiefly in pathologic conditions of the anterior segment of the eye, particularly staphylococcic infections. It is given both systemically and locally—systemically as 40,000 u. every two to four hours, or 300,000

35. Dekking, H. M.: Use of Hyaluronidase in Some Corneal Affections, Ophthalmologica **119**:63-64 (Jan.) 1950.

36. Offret, G., and Chauvet, P.: Contribution to the Study and Treatment of Vascularization of the Cornea: IV. Therapy, Arch. opht. **10**:593-612, 1950.

37. Parisi, P. J.: Ocular Tuberculosis Treated with Streptomycin: Report of 14 Cases, Am. J. Ophth. **34**:393-401 (March) 1951.

38. Thierry, I., and Hevia, R.: Ocular Tuberculosis and Streptomycin, Arch. Chilenos de oft. **5**:617-623 (Jan.-Dec.) 1948.

39. Woods, A. C.; Wood, R. M., and Naquin, H. A.: Studies in Experimental Ocular Tuberculosis, A. M. A. Arch. Ophth. **45**:546-554 (May) 1951.

40. Fried, J. J.: Recent Advances in Ocular Therapy, Am. J. Ophth. **33**:1881-1890 (Dec.) 1950.

u. daily, and locally as ointment (1,000 to 5,000 u. per gram, or 100,000 u. per gram), as drops (30,000 u. per fluid ounce [30 cc.] of saline solution), or as subconjunctival injection (1 cc. containing 5,000 u., with some procaine hydrochloride and with a wetting agent). The sulfonamide drugs are sulfapyridine, sulfathiazole, sulfadiazine, and mixed sulfonamide drugs (disulfonols, trisulfonols); sulfadiazine is the least toxic and is therefore the sulfonamide of choice for either local or systemic use. It is most effective against streptococcic infections and is given systemically as 2 gm. at the start and then as 0.5 gm. every four hours for 6 to 12 days and locally as a 5% ophthalmic ointment or solution. Streptomycin is particularly useful against Pseudomonas aeruginosa (Bacillus pyocyaneus) and certain Gram-negative bacteria, dihydrostreptomycin hydrochloride being less toxic and less likely to produce toxic optic neuritis. The dose is 500,000 to 1,000,000 γ daily, to a total of 25 to 30 gm.; locally it can be used as a 5% ophthalmic ointment. Bacitracin is one of the newer antibiotics and has been found to be effective in pathologic conditions of the external eye caused by Gram-positive organisms. It is used topically only, as a solution containing 500 u. per cubic centimeter of isotonic sterile sodium chloride or as an ophthalmic ointment containing 500 u. per gram in wool fat U. S. P.-cold cream base. Aureomycin is good in treatment of virus infections; systemically it may be given as 250-mg. tablets orally every two to three hours for 6 to 12 days, and locally as eye drops of a 0.5% solution (25 mg. of aureomycin in 5 cc. distilled water with 62.5 mg. sodium chloride and 25 mg. sodium borate) or as a 0.1% ophthalmic ointment. In the same category is terramycin [41]; it can be used in a 0.5% alkaline solution or in an acid solution in methylcellulose, the latter apparently being more effective. This drug has been found useful against most of the micro-organisms and some of the viruses causing external ocular infections.

A preliminary note on the use of paraaminobenzoic acid was submitted by Scuderi.[42] He reports that this drug has a wide spectrum of pharmacologic action, including, among others, antibacterial, antirickettsial, antirheumatic, and anti-allergic effects, and a synergistic action with penicillin and streptomycin. His data are not yet confirmed, however.

B. Cortisone and Corticotropin: The use of cortisone and corticotropin has increased greatly during the past year; consequently, reports on their actions and effects are beginning to appear. Not all reported results are favorable; Thygeson, Geller, and Schwartz [43] tried cortisone (intramuscularly, subconjunctivally, and topically) in experimental herpes-simplex keratitis in the rabbit, without amelioration of the lesions. On the other hand, Steffensen and others,[44] at Henry Ford Hospital, report on their topical use of cortisone for various diseases of the anterior segment and state that there was alleviation of symptoms in 3 to 72 hours, with eventual cure. The various diseases thus treated were (*a*) nonspecific keratitis

41. Town, A. E.: Ophthalmic Use of Terramycin, Am. J. Ophth. **34**:723-726 (May, Pt. 1) 1951.

42. Scuderi, G.: Para-Aminobenzoic Acid in Ophthalmology; Preliminary Note, Boll. ocul. **29**:654-655 (Oct.) 1950.

43. Thygeson, P.; Geller, H. O., and Schwartz, A.: Effect of Cortisone on Experimental Herpes-Simplex Keratitis of the Rabbit, Am. J. Ophth. **34**:885-887 (June) 1951.

44. Steffensen, E. H., and others: Topical Cortisone in the Treatment of Anterior-Segment Eye Disease, Am. J. Ophth. **34**:345-346 (March) 1951.

profunda, (*b*) superficial keratitis, (*c*) postoperative striate keratitis, (*d*) phlyctenular keratoconjunctivitis, (*e*) tuberculous keratitis, (*f*) recurrent acne-rosacea keratitis, (*g*) dendritic ulcer, (*h*) acute marginal ulcer, (*i*) trophic ulcer, and (*j*) Fuchs's epithelial dystrophy. Cortisone was used topically in these cases; the preparation was made by diluting 1 volume of saline suspension of cortisone with 4 volumes of isotonic sodium chloride solution, and it was first instilled in the eye every 30 to 60 minutes while the patient was awake, the interval being increased with improvement. Woods [45] has given a most useful summary of his observations on the use of cortisone and corticotropin; they are as follows:

1. ACTH [Corticotropin] and cortisone given systemically have a profound effect in the control of ocular inflammation and exudation in the ocular humors.

2. Cortisone administered locally in the conjunctival sac has a similar effect on external ocular inflammation, the beneficial effects being uniformly observed after 2 to 4 days of intensive therapy.

3. The optimum method of treatment . . . has yet to be determined.

4. Experimental studies confirm the clinical observations. ACTH, administered systemically, and cortisone, administered systemically and topically, block in the eye the inflammatory and exudative phases of the anaphylactic protein reaction, . . . and of the focal reaction produced by the subcutaneous injection of tuberculin.

5. Both ACTH and cortisone given systemically appear to depress temporarily the cutaneous reactions incident to drug allergy and bacterial and tuberculin hypersensitivity. . . .

7. Although ACTH and cortisone block the inflammatory and exudative phases of the anaphylactic and allergic reaction, the evidence so far at hand indicates that they have no effect on the underlying hypersensitive state.

8. The mode of action of ACTH and cortisone is as yet undetermined. It seems probable that they act in some way on the mesenchymal tissues.

Most writers agree that these two drugs are particularly useful in diseases the pathogenesis of which is of an allergic nature.

C. Irradiation: Mention must be made of the increasing usefulness of irradiation in ophthalmology. This irradiation may assume such varied forms as roentgen therapy, beta irradiation, and use of Grenz rays. Roentgen therapy has shown its effectiveness in herpetic keratitis, bullous keratitis, interstitial keratitis, sclerosing keratitis, and phlyctenular keratitis. Strazzi [46] reports the work of the clinic in Bologna; here, the factors were 2 ma., 170 to 180 kv., 0.5 mm. Zn and 3 mm. Al filtration, 33 cm. skin-target distance, 60 to 80 r per session, and five or six sessions every five to six days. Fedrizzi and Ferri [47] report the work of the clinic in Pavia; there the factors were 30 to 40 r every second day up to 180 to 320 r; 25 cm. target-skin distance; 55 kv., and 0.5 mm. Al filtration.

Beta irradiation is excellent for the treatment of certain lesions of the anterior ocular segment. Three types of applicators are now being used. Iliff [48] states that new corneal vascularization can be controlled by beta therapy and existing vessels

45. Woods, A. C.: Clinical and Experimental Observation on the Use of ACTH and Cortisone in Ocular Inflammatory Disease, Am. J. Ophth. **33**:1325-1349 (Sept.) 1950.

46. Strazzi, A.: X-ray Therapy of the Cornea, Atti 37 Cong. Soc. oftal. ital. **10**:578, 1948.

47. Fedrizzi, G., and Ferri, L.: Roentgen Therapy of Superficial Ocular Lesions, Atti d. 37 Cong. Soc. oftal. ital. **10**:435-439, 1948.

48. Iliff, C. E.: Present Status of Beta Irradiation in Ophthalmology, Proceedings of 16th International Congress of Ophthalmology, July, 1950, Vol. 2, pp. 1357-1362.

occluded; Ruedemann [49] finds that marginal epithelial overgrowth, corneal nebulae, leucoma, dermoids of the cornea, and active interstitial keratitis respond to beta irradiation.

The latest type of radiation to be added to the forms used in irradiation is the soft Grenz ray; these x-rays are the least penetrating, with a wave length of 1.5 to 2.0 A., and they exert influence on the most superficial layers of the tissues. The work done to date has been on rabbits,[50] whose limit of tolerance was 4,000 r, given as a single dose. This should be ideal for use on the cornea.

## SCLERA

Again, the sclera has received scant attention in the literature. The chief affections of the sclera are allergic or traumatic; in the former classification fall rheumatoid and tuberculous nodules of the sclera. Episcleritis and scleritis are commonly seen with disease of the joints, especially rheumatoid arthritis. The disease takes the form of nodules. The most efficacious therapeutic agent recently found is cortisone. Used locally, it causes amelioration of the symptoms; on cessation of administration, a definite exacerbation is noted.[51] Intramuscular administration causes disappearance of the nodular formations.

Tuberculous nodules of the sclera are usually solitary and are secondary, with hematogenous origin from a tuberculous focus, which is only infrequently an active pulmonary lesion. Tuberculoma of the sclera is in itself a rare disease; the lesion usually appears in the circumcorneal area and tends to involve the cornea and underlying uveal tissue. In the past, treatment has been limited to general care plus excision of the lesion, followed with tuberculin therapy. Maiden [52] advocates administration of streptomycin systemically, with local use of atropine and excision. Streptomycin, 2 gm., is given daily in divided doses until the lesion subsides.

Swan [53] has recently published work on contemporary concepts of scleral disease. He gives, first, a histologic description of the sclera, stating that rather than being a "fibrous" coat, it is in reality a collagenous tissue, which responds as such to inflammation. When an acutely destructive injury occurs (an operative incision or the focal destruction of diathermy) proliferative scleritis results, and the edges of the penetrating wound usually tend to retract. The defect is filled in by a proliferation of mesenchymal tissue from the episclera and choroid; the process is like healing by secondary intention. This type of healing is important in cataract extraction, since, if the incision is behind the limbus, there may occur the complication of fibroblastic ingrowth; this must be differentiated from epithelial downgrowth. The fibroblastic ingrowth extends down the posterior surface of the cornea;

49. Ruedemann, A. D.: Beta Ray Uses in Ophthalmology, Wisconsin M. J. **49**:581-584 (July) 1950.

50. Katzin, H. M., and Okrainetz, C.: Grenz-Ray Irradiation of Vascularized Rabbits' Corneas, Am. J. Ophth. **33**:1904-1906 (Dec.) 1950.

51. Mundy, W. L.; Howard, R. M.; Stillman, P. H., and Bevans, M.: Cortisone Therapy in Case of Rheumatoid Nodules of the Eye in Chronic Rheumatoid Arthritis, A. M. A. Arch. Ophth. **45**:531-538 (May) 1951.

52. Maiden, S. D.: Solitary Tuberculoma of the Sclera, in a Patient with Multiple Advanced Systemic Lesions; Treated with Streptomycin, Am. J. Ophth. **34**:387-392 (March) 1951.

53. Swan, K. C.: Some Contemporary Concepts of Scleral Disease, A. M. A. Arch. Ophth. **45**:630-644 (June) 1951.

the surface is gray and looks like woven cloth. It is usually self-limited but sometimes must be checked by ionizing irradiation or local application of cortisone.

Exudative scleritis can be either suppurative or granulomatous. The former occurs when injury incites intense infiltration of the tissue with polymorphonuclear leukocytes to form an abscess. The latter type is characterized by infiltration of the sclera with large masses of inflammatory cells; the infiltration and inflammatory edema result in irreparable damage to the fibers. It usually involves the anterior-chamber segment in the region of the perforating vessels. Syphilis, tuberculosis, and sarcoidosis are the common etiological agents, but it may also result from chemical and physical irritants or from a hypersensitivity factor, e. g., erythema nodosum, rheumatoid arthritis, or disseminated lupus erythematosus. Lesions which can be found where hypersensitivity plays an important role are scleromalacia perforans (associated with arthritis), phlyctenular disease, and hyaline plaques (the result of a disturbance in metabolism). Various scleropathies, a term used by Swan to include such lesions as staphyloma, are also described.

## SURGERY

As keratoplasty becomes a commoner operative procedure and an increasingly important adjunct to the field of ophthalmic surgery, more information rapidly becomes available. This is evidenced by the fact that experiments on keratoplastic procedures are being conducted and reported. Ferrata and Morpurgo [54] and Ferrata, Morpurgo, and Dugnani [55] have made important contributions in this field. Sections were made of eyes with experimental keratoplasties, showing normal healing, as well as operative complications. The belief of these authors is that in successful cases the intercellular structures of the graft are accepted and persist; the epithelium and endothelium are partially replaced by migration of the cells of the recipient cornea. The stromal cells partially degenerate; the newly formed keratoblasts are of doubtful origin. In cloudy grafts there are shown massive degeneration and replacement of the transplanted tissues. Occasionally, newly formed connective tissue grows into the graft. These authors also noted that the nerves of the transplant begin to degenerate within two weeks after operation. There is reinnervation of the graft from the nerves of the host's cornea, first apparent at the end of the second month. Regeneration of nerves was seen in both opaque and clear grafts.

According to Paufique,[56] the indications for lamellar keratoplasty are (1) corneal dystrophies (lattice and nodular dystrophy); (2) leucomas from infective keratitis, when superficial and well vascularized; (3) traumatic leucomas, especially after burns of the cornea; (4) aphakia, when there is vitreous in the anterior chamber; (5) one-eyedness, with poor visual acuity, and (6) in children, nervous patients, or patients with nystagmus.

Indications for therapeutic lamellar grafts are (1) neurotropic virus keratitis, recurrent herpetic keratitis, or disciform keratitis; (2) prolonged corneal ulceration, even with hypopyon, or corneal abscess; (3) interstitial keratitis (Stage 1 or 2);

54. Ferrata, L., and Morpurgo, F.: Experimental Research in Keratoplasty, Gior. ital. oftal. **3**:188-203 (June) 1950.

55. Ferrata, L.; Morpurgo, F., and Dugnani, E.: Experimental Keratoplasty, Gior. ital. oftal. **3**:366-375 (Sept.-Oct.) 1950.

56. Paufique, L.: Lamellar Keratoplasty: Present-Day Uses, Proceedings 16th International Congress of Ophthalmology, July, 1950, Vol. 2, pp. 1037-1043.

(4) recurrent pterygium; (5) keratitis following injury, cataract extraction, or glaucoma, or (6) diseased graft after perforating keratoplasty.

Therapeutic lamellar grafts are used to stop the development of progressive corneal disease, particularly if perforation is feared. A combination of lamellar and perforating keratoplasty was tried by Franceschetti and Doret [57]; they found that, regardless of the technique employed, the results were uniformly unsatisfactory. Both human and plastic grafts were eliminated; the authors suggest a "mushroom" graft, for cutting of which their special instrument is designed. Bock [58] also describes a new instrument for use in lamellar keratoplasty; it consists of half a razor blade with a thin bar a short distance in front of and above its cutting edge. The bar presses the cornea flat, while the blade, immediately following it, produces an even, smooth cut.

Closely related to the study of Paufique on indications is Sourdille's [59] study of techniques of lamellar keratoplasty. The technique employed in total lamellar keratoplasty, which is reserved for very damaged corneas, is described as follows: The desired area is outlined by a trephination of 10 to 11 mm., the depth not exceeding half the thickness of the cornea; the corneal flap is resected with Desmarres' scarifying knife, assuring rigorous hemostasis; the graft is cut, placed, and fixed with an edge-to-edge suture; the eyelids are temporarily sutured. Barraquer Moner [60] recommends edge-to-edge sutures; he has modified the Vogt needle by making a sharper point and a longer cutting edge. The suture must pass through the anterior half of the corneal stroma, and the stitches should be placed radially. The edge-to-edge suture successfully resists the intraocular tension. The partial lamellar keratoplasty, according to Sourdille, is applied to less severe lesions, and the varieties of technique are numerous:

(*a*) Circular central keratoplasty

(*b*) Peripheral keratoplasty

(1) Annular type, either complete (extensive Mooren ulcer) or incomplete (smaller Mooren ulcer, extensive or recurrent pterygium, trophic graft for interstitial keratitis)

(2) Segmentary type (recent pterygium)

(3) Crescent-moon type (lagophthalmos, inferior leucoma following exophthalmos)

Some technical considerations (concerning trephines, center of pupil, application of trephine over center of pupil, and accurate insertion of sutures) are discussed by Thomas.[61] Castroviejo,[62] stimulated by the results published by Paufique, Sourdille, and Offret, tried both total and partial lamellar types of keratoplasty in this country and published his results. He outlines the following indications:

57. Franceschetti, A., and Doret, M.: Combined Lamellar and Perforating Keratoplasty, Proceedings 16th International Congress of Ophthalmology, July, 1950, Vol. 2, pp. 1050-1060.

58. Bock, R. H.: A New Instrument for Lamellar Keratoplasty, Arch. Ophth. **44**:293-299 (Aug.) 1950.

59. Sourdille, G. P.: Techniques of Lamellar Keratoplasties, Proceedings 16th International Congress of Ophthalmology, July, 1950, Vol. 2, pp. 1044-1049.

60. Barraquer Moner, J.: Keratoplasty: Edge-to-Edge Sutures, Proceedings 16th International Congress of Ophthalmology, July, 1950, Vol. 2, pp. 999-1004.

61. Thomas, J. W. T.: Keratoplasty: Some Technical Considerations, Proceedings 16th International Congress of Ophthalmology, July, 1950, Vol. 2, pp. 993-998.

62. Castroviejo, R.: Lamellar Keratoplasty: Technique and Results; Comparative Study with Penetrating Keratoplastics and Keratectomies, Am. J. Ophth. **33**:1851-1862 (Dec.) 1950.

1. For optical keratoplasty—superficial lesions. The best results are obtained in corneal, especially nodular, dystrophies, Groenow and Haab-Dimmer types; fatty corneal dystrophy and primary chronic corneal edema (Fuchs's dystrophy); sequelae of infectious keratitis; superficial opacities after ulcer, and mustard-gas keratitis.

2. As a preliminary to penetrating keratoplasty—very dense leukomas.

3. As therapeutic lamellar transplants—trophic action. Such transplants are indicated in all forms of chronic or recurring keratitis for which the usual methods of treatment have failed. The best results are obtained in keratitis due to neurotropic viruses, especially herpetic keratitis and keratitis of herpes zoster; in syphilitic and tuberculous keratitis, and in the keratitides of undetermined origin. Castroviejo also points out the differences between his methods and those of Paufique, Sourdille, and Offret.

Acrylic material is being widely used in transplantation surgery. The corneal graft must be fixed to prevent tilting and opacification; an acrylic disk splint with four flanges anchored to the cornea can be used to do this. The splint is transparent and permits observation of the graft throughout the entire procedure. Acrylic implants are also used and found to be well tolerated and technically excellent. Shotter [63] describes another method of fixing the graft in keratoplasty; after the trephine has been driven through two-thirds of the thickness of the donor cornea, two fine stitches on eyeless needles are passed through two ends of any diameter of the outlined graft. The needles and stitches are next passed through the trephine with a hook, after which the trephine incision is completed and the graft is transferred to the recipient eye and fixed by passing the same needles and stitches through the host's cornea.

Pertinent to this work, and undoubtedly of great interest to the surgeon, is the report of Castroviejo [64] on his unusual cases, particularly those in which retransplants are required. He has found the lamellar type of keratoplasty very useful in the treatment of corneal opacities and deformities which were formerly treated only by partial penetrating keratoplasty. This article deals chiefly with the application of keratoplasty to eyes with an unfavorable prognosis; these are: (1) eyes requiring retransplants, the good results showing that keratoplasty may be successful after one or more operations have resulted in cloudy or opaque grafts; (2) eyes, formerly considered unfavorable, which might benefit by corneal transplantation if they were properly prepared by preliminary treatment, e. g., irradiation for obliteration of corneal vascularization. Cases are reported in which eventually a clear graft was obtained after several failures.

Three hundred cases of corneal grafting are reported by Löhlein [65]; he describes his technique (partial perforating keratoplasty) and indicates the groups into which the cases fall, as follows: chemical burns (46 cases); foreign-body injuries (23 cases); scarring from gonorrheal blennorrhea (7 cases); scarring from measles,

63. Shotter, L.: A New Method of Fixing the Graft in Keratoplasty, Vestnik. oftal. **29**:38-39 (Nov.-Dec.) 1950.

64. Castroviejo, R.: Keratoplasty: Retransplants; Unusual Cases, Proceedings 16th International Congress of Ophthalmology, July, 1950, Vol. 2, pp. 1005-1015.

65. Löhlein, W.: Experiences with Corneal Grafting, von Graefes Arch. Ophth. **151**:1-45, 1950.

scarlet fever, vaccinia keratitis, keratomalacia, and keratitis urica (14 cases); scarring from tuberculosis (16 cases); keratic precipitates (38 cases); leucoma following corneal ulceration (16 cases); scarring of unknown origin (23 cases); heredodegenerations (10 cases); keratoconus (12 cases); rosacea keratitis (13 cases); fistulating conditions (15 cases); ulcus serpens and corneal abscess (13 cases); herpetic infections (6 cases). Hypopyon keratitis was treated with corneal graft by Gallenga,[66] as was chronic dendritic ulceration by Foster.[67]

The changes in the graft, as well as in the recipient cornea, are of great interest, since knowledge of them may be important in determining the prognosis in a prospective case. Joseph and Offret[68] studied postoperative bioclinical changes after corneal grafting in 80 cases. When there was active corneal disease, there was an immediate and definite effect on pain. The effect on the disease was variable; in some cases cure was complete, while in others the disease continued to develop. When the grafts were made for scars of the cornea, two effects were found: one on the recipient cornea and one on the donor graft. The recipient cornea showed improvement of epithelial lesions but no change in the neighboring stromal opacities; the graft may also act on certain opacities of the endothelium and Descemet's membrane. There is also a temporary increase of vessels in the recipient cornea. Without the host, the graft will die after the 14th day. Opacification of the graft is a common and dread consequence. Carapanca[69] believes that this opacification results many times from failure to respect the optical and mechanical zones of the cornea, of which he estimates there are five (pupillary zone divided into 6.11, 2.17, 90, 0.95, and 0.64%, of the thickness of the cornea).

The consensus, however, is that opacification is due chiefly to an allergic reaction between the donor and the host cornea. Klíma[70] injected crushed corneal extract into the cornea of rabbits and found allergic reactions in cases in which opacification developed. Maumenee[71] supports the belief in an allergic origin and states that the type of reaction is dependent on the biologic specificity of individual animals of the same species. The cases in which opacification developed by the 10th postoperative day belong to this group. Late causes of opacification are (*a*) defects in donor material, (*b*) poor apposition of the donor-recipient tissues, (*c*) postoperative pyogenic infections, (*d*) extension of a disease process, (*e*) foci of lymphocytes in old corneal scars, and (*f*) destruction of a hypothetical enzyme system in the cornea.

Other types of corneal surgery have received little attention recently. This is probably due to the fact that those described have been encompassed by more

66. Gallenga, R.: Corneal Transplants in Kerato-Hypopyon, Rass. ital. ottal. **19**:73-76 (March-April) 1950.

67. Foster, J.: Case of Chronic Dendritic Ulceration Treated by a Lamellar Corneal Graft, North England Ophth. Soc. **70**:59-91, 1950.

68. Joseph, E., and Offret, G.: Post-Operative Bioclinical Observations After Corneal Grafting, Proceedings 16th International Congress of Ophthalmology, July, 1950, Vol. 2, pp. 1016-1023.

69. Carapanca, M.: A New Biologic Approach in Partial Keratoplasty: Respect for the Bioarchitecture of the Cornea, Arch. opht. **10**:51-57, 1950.

70. Klíma, M.: Antigenic Properties of the Corneal Protein, Sbor. lék. **51**:1-34, 1949.

71. Maumenee, A. E.: The Influence of Donor-Recipient Sensitization on Corneal Grafts, Am. J. Ophth. **34**:142-152 (May, Pt. 2) 1951.

modern approaches to these same problems. Miklos [72] writes of healing a serpiginous ulcer of the cornea by total conjunctival hooding; he describes his method in detail and reports success. Jayle and Dor [73] have tried perilimbic annular grafting of buccal mucous membrane; they prefer this method for treating various ocular diseases for which corneal grafting is often indicated. Their technique is also described. The membrane lining an eggshell was used by Oxilia and Carlevaro [74] in the repair of corneal wounds to obviate the use of corneal sutures, conjunctival flaps, and tarsorrhaphy. These authors believe that perfect closure of the wound is obtained, and this membrane is well tolerated. Its transparency is useful as a window through which to watch the progress. Interstitial keratitis was treated in China with corneoscleral trephination by Wu [75]; she reports excellent results in the four eyes thus treated.

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# Correspondence

## SYMPATHETIC OPHTHALMIA

*To the Editor:*—I feel compelled to write to you concerning a case of proved sympathetic ophthalmia which was successfully treated by enucleation of the exciting eye and simultaneous administration of average doses of aureomycin by mouth. I am prompted to do this because of your editorial in the November 1951 issue of the A. M. A. ARCHIVES, page 488. This clinical experience, with accumulated statistics coming to your attention since the advent of antibiotics, may be of value.

F. M., a white woman aged 63, was operated on for removal of a senile cataract. Preoperative visual acuity was limited to counting fingers at 2 ft. (60 cm.) in the right eye and was 20/400— in the left eye. The intraocular tension was normal; the conjunctivas were clear, and the lids and lacrimal sacs clean. Intracapsular extraction was performed on the right eye on April 25, 1949, with a round pupil and placement of two McLean sutures; there were no surgical complications. The immediate postoperative course was stormy, with uncontrollable nausea and violent vomiting. Before the patient could be quieted with medication, she said something "popped" in her eye. Later the eye was examined, and it was ascertained that the patient had an expulsive choroidal hemorrhage. Sight, as tested by projection of light, was absent; permission for further operation and, later, for enucleation was denied. Forty days after operation, during which period the fellow eye was watched closely, sympathetic uveitis appeared.

The exciting eye was removed. Streptomycin, 0.5 gm. twice a day was given intramuscularly, and aureomycin hydrochloride, 250 mg., was given orally every six hours. In addition, a course of nonspecific typhoid foreign protein therapy was given, and always the sympathizing eye was under atropine cycloplegia. Dr. J. S. Friedenwald examined the specimen and later saw the patient in consultation, suggesting tanning exposures to ultraviolet rays.

Two weeks after the beginning of streptomycin therapy, it was stopped because of hearing difficulties. By Aug. 5, the pupil was widely dilated, with a few remaining keratic precipitates and Koeppe nodules. Since these had a washed-out appearance and the patient was showing gastrointestinal symptoms, probably due to aureomycin, we decreased the dose to 250 mg. twice a day. Between Oct. 4 and Oct. 25, 1949, keratic precipitates and Koeppe nodules, with aqueous flare, reappeared in small sizes and numbers. However, by reverting to administration of 250 mg. of aureomycin every six hours, the active process was checked by Nov. 5, 1949. In January, 1950, the eye was white; keratic precipitates and Koeppe nodules disappeared. The pupil was dilated widely, with only two small pigmented, marginal synechiae, pinpoint in size, which did not deform the pupil. There was no degenerative change in the cornea; the lens showed no increase in cortical or nuclear opacities. The retina, seen poorly, showed a normal disk and macula, and there was no vascular abnormality. Visual acuity was limited to counting fingers at 5 ft. (1.5 meters), aided somewhat with a —4.50 D. lens.

During the next two months the number of capsules of aureomycin hydrochloride, 250 mg., was decreased one at a time from four to one, and their use was finally stopped entirely. Since then, the eye has been clear; the patient is able to discriminate colors, and she reads typewritten print with a magnifying glass for a short time. Once atropine allergy developed, which cleared when the cycloplegic was changed to scopolamine. No cortisone has been used. The intraocular pressure remains at 25 mm. Hg, as measured with a McLean tonometer. The patient has taken good care of her general health, receiving injections of vitamin preparations.

Rightly or wrongly, I cannot but believe that aureomycin has had a happy effect in this case. After reading Dr. Schreck's article and your editorial, I felt obliged to extract this information from my files.

KARL B. BENKWITH, M.D.,
Montgomery, Ala.

# News and Notes

EDITED BY DR. W. L. BENEDICT

## PERSONAL NEWS

**Leslie Dana Medal Awarded.**—The St. Louis Society for the Blind has announced the award of the Leslie Dana Medal for the Prevention of Blindness to Dr. E. V. L. Brown, Chicago. A dinner honoring Dr. Brown was given on Friday, Dec. 7, in St. Louis by the St. Louis Society for the Blind, in cooperation with the National Society for the Prevention of Blindness and the Association for Research in Ophthalmology.

## SOCIETY NEWS

**Indianapolis Ophthalmological and Oto-Laryngological Society.**—The officers of the Indianapolis Ophthalmological and Oto-Laryngological Society for 1951-1952 are as follows: Marlow W. Manion, M.D., president; Robert M. Dearmin, M.D., vice-president; Lewis E. Morrison, M.D., Secretary-Treasurer.

## GENERAL NEWS

**Combat Blindness.**—The National Council to Combat Blindness, in accordance with its program of supporting ophthalmologic research, is now welcoming applications for its third annual grant-in-aid awards, to be made in June, 1952. This announcement will be of interest to the personnel in medical schools and other institutions concerned with ophthalmologic research. Applications to be considered for this group of awards should be in the office of the Council no later than April 15, 1952. Application forms may be obtained by addressing the Secretary, National Council to Combat Blindness, 1186 Broadway, New York 1.

The organization's Committee on Research, consisting of Dr. Phillips Thygeson, University of California School of Medicine, San Francisco, Calif., chairman; Dr. Alson E. Braley, State University of Iowa College of Medicine, Iowa City; Dr. Dan M. Gordon, Cornell University Medical College, New York; Dr. Stuart Mudd, University of Pennsylvania School of Medicine, Philadelphia; Dr. Charles A. Perera, Columbia University College of Physicians and Surgeons, New York; and Dr. Samuel L. Saltzman, New York Medical College, Flower and Fifth Avenue Hospitals, awarded the following grants-in-aid in 1951:

Retina Foundation, Massachusetts Eye and Ear Infirmary, Boston, $5,000, Dr. Endre A. Balazs, studies on acid mucopolysaccharides in the vitreous body, their role in the composition of the vitreous body, and their relation to the proteins in the vitreous gel; University Hospitals, State University of Iowa, Iowa City, $2,700, Dr. Hermann M. Burian, study of the electrical response of retina and brain in patients with amblyopia ex anopsia and suppression; The Post-Graduate Medical School of New York University-Bellevue Medical Center, New York, $1,296, Dr. Helena Fedukowicz, attempts to grow the virus of inclusion conjunctivitis, follicular conjunctivitis, and folliculosis; New York Eye and Ear Infirmary, New York, $1,600, Dr. Louis J. Girard, focal infection clinic; State University of Iowa College of Medicine, Iowa City, $1,200, Dr. Ralph C. Janes, a study of the incidence and basic cause of cataract, hemorrhages, and degenerative changes in the retina and optic nerve of animals with alloxan diabetes; University of California School of Medicine, San Francisco, Calif., $1,200, Dr. Samuel J. Kimura, research on the relation of uveitis to bacterial allergy; University of Pennsylvania School of Medicine, Philadelphia, $2,500, Dr. H. P. Kirber, study of virus infections of ocular tissues grafted onto the chorioallantoic membrane of the chick embryo; Government Hospital, Haifa, Israel, $2,500, Dr. I. C. Michaelson, study of factors affecting new vessel growth into the cornea; Indiana University School of Medicine, Indianapolis, $1,500, Dr. T. F. Schlaegel Jr., studies on the mechanism of development of contralateral granulomatous uveitis from the use of horse serum in rabbits; New York Hospital-Cornell Medical Center, New York, $750, Dr. Edward Tolstoi, studies on retinopathy in diabetes; New York Medical Col-

lege, Flower and Fifth Avenue Hospitals, New York, $466, Dr. Charles Haig, continuation of study on regional light sensitivity of the retina.

Dr. James H. Allen, of Tulane University of Louisiana School of Medicine, New Orleans, has joined the organization's Committee on Research.

**Fourth Annual Clinical Conference, Chicago Ophthalmological Society.**—The Chicago Ophthalmological Society has announced that the Fourth Annual Clinical Conference is to be held Feb. 21-23, 1952, at the Drake Hotel. The first day will be devoted to surgical clinics at various Chicago hospitals. On the following days there will be symposiums on cataract surgery and recent advances in therapeutics, followed by round-table luncheons. In addition, there will be lectures on neuro-ophthalmology, ocular pharmacology, nystagmus, provocative tests in glaucoma, and the secretion of aqueous humor. Participants include Dr. Alson E. Braley, Iowa City; Drs. Justin M. Donegan, Joseph S. Haas, Peter C. Kronfeld, James E. Lebensohn, Samuel J. Meyer, Maurice D. Pearlman, and Derrick Vail, Chicago; Dr. Irving H. Leopold, Philadelphia; Drs. John M. McLean and Joseph A. C. Wadsworth, New York, and Dr. C. Wilbur Rucker, Rochester, Minn.

Further information concerning the conference may be obtained from Miss Maud Fairbairn, 8 W. Oak St., Chicago 10.

The conference will be followed by a banquet and the eighth annual Sanford R. Gifford Memorial Lecture, to be presented by Dr. Alson E. Braley, professor and head of the department of ophthalmology, State University of Iowa College of Medicine. Dr. Braley will speak on "Experimental Studies of the Herpes Simplex Virus."

**Grants from the National Society for the Prevention of Blindness.**—Limited funds are available for grants for basic laboratory or clinical research projects that may add to understanding of the etiology of blinding disease of the eyes or improve methods of diagnosis, treatment, or prevention. To be considered, applications should be received before April 1, 1952. Forms may be obtained from the National Society for the Prevention of Blindness Research Committee, 1790 Broadway, New York 19.

**Postgraduate Conference in Ophthalmology, University of Michigan Medical School.**—The Department of Postgraduate Medicine of the University of Michigan Medical School announces the annual conference in ophthalmology for qualified physicians, April 21, 22, and 23, 1952, to be given at the Horace H. Rackham Graduate School Building, Ann Arbor, Mich., under the direction of the department of ophthalmology at the University of Michigan Medical School.

Complete program and details will be mailed upon request addressed to Howard H. Cummings, M.D., chairman, Department of Postgraduate Medicine, University Hospital, Ann Arbor, Mich.

**Glaucoma Laboratory, University of Toronto.**—The Department of Ophthalmology of the University of Toronto has started a glaucoma laboratory in the Banting Institute, under the direction of Dr. T. H. Hodgson. This has been made possible through a grant from the Ontario Provincial Department of Health with funds provided by federal grant. The laboratory will undertake basic studies on glaucoma in conjunction with the glaucoma clinic service. The staff members include Dr. W. P. Callahan, Dr. R. K. MacDonald, and Miss S. Henninghausen, technician.

**New Ophthalmology Building, Toronto General Hospital.**—Plans for construction of a new ophthalmology building has been announced by the Board of Trustees of the Toronto General Hospital as part of a $14,000,000 expansion program. This building will be a five-story structure, containing 75 beds, outpatient eye clinics, private offices, laboratories, postgraduate and undergraduate lecture theaters, research laboratories, eye operating rooms, etc. It is estimated that the building will cost $1,350,000.

# Book Reviews

**The Normal Cerebral Angiogram.** By Arthur Ecker, M.D., Ph.D. Price, $6.50. Pp. 216, with 147 illustrations. Charles C Thomas, Publisher, 301-327 E. Lawrence Ave., Springfield, Ill., 1951.

The technique of angiography has been so improved and simplified that visualization of the cerebral blood vessels is now a routine in all cases in which an organic intracranial disorder, such a tumor, aneurysm, or hematoma, is suspected. Ophthalmologists are particularly concerned with the cerebral blood vessels and will find this book of considerable value. The knowledge of normal angiographic anatomy is a prerequisite to the understanding of angiograms and pathologic conditions, and this work is designed primarily to provide a background for the interpretation of abnormal cerebral angiograms.

The book is divided into two parts. Part I deals with the technique and complications. The portion on the latter should be read by all ophthalmologists before referring patients for this procedure. Part II concerns angiographic anatomy and illustrates the normal cerebral angiograms and the common artifacts. Of special interest to ophthalmologists are the following topics:

1. A method of surveying angiographically both internal carotid arteries and the basilar artery and their branches (of special value in cases of spontaneous subarachnoid hemorrhage or increased intracranial pressure without localizing signs).

2. Carotid arteriograms and venograms in a projection used to demonstrate the optic canal (of particular interest in cases of primary optic atrophy of unknown cause, which may be due to arteriosclerosis of the internal carotid, ophthalmic, or anterior cerebral artery).

The illustrations are excellent, and the text is clear and concise.

**Textbook of Refraction.** By Edwin F. Tait, M.D. Price, $7.50. Pp. 418. W. B. Saunders Company, 218 W. Washington Sq., Philadelphia 5, 1951.

In contrast to the several other books recently published on optics and refraction, this book is written specifically for the clinician. The text is concerned with method more than with answering the question "Why?" Perhaps it lacks, therefore, a little of the thoroughness one usually expects from a textbook. The author presupposes a knowledge of the fundamentals of optics and of formation of images by lenses.

The organization of the book was at first a little confusing to the reviewer, because significant topics are briefly considered early in the text only to be treated later at greater length. This organization may lead to a more thorough understanding of the subject matter. The book is roughly divided into three sections. The first gives general considerations to refractive errors, ocular reflexes, and anomalies of accommodation. The second section, which constitutes by far the major part of the book, deals with the problems of binocular coordination. Herein lies Dr. Tait's special interest, and it is in this part that the book provides its essential contribution. The important topics are the anatomy and physiology of the ocular muscles, the convergence reflexes, heterophorias, the Duane-White classification of anomalies of convergence, prism vergences, relationships between phorias and prism vergences for comfortable binocular vision, fusion exercises, etc. The third part of the book deals specifically with methods of refraction and with the procedure in arriving at the best correction for refractive errors and heterophorias. In this part are discussed the importance of the patient's history, the determination of visual acuity, the use and value of the keratometer, skiametry (retinoscopy), dynamic skiametry, the use of cycloplegics, methods of subjective determination of refractive errors, special problems in aphakia, subnormal vision,

contact lenses, and orthoptics. The book ends with a rather long, step-by-step description of a system of refraction and the interpretation of the data obtained in order to arrive at the best refractive correction. The average busy ophthalmologist will consider this outline far too long and, on the whole, unnecessary.

The author apparently uses cycloplegics with trepidation, and then rarely for patients more than 35 years of age. He suggests, furthermore, the use of an artificial pupil when the refractive error is determined with the eye under cycloplegia, in order to eliminate the unnatural aberrations inherent with the enlarged pupil. Dynamic skiametry is discussed at some length because the author is of the opinion that with this method more precise results can be obtained than with static skiametry, provided the exact amount of accommodation being used at the moment can be determined. His procedure for finding this is explained.

The format and reproductions of the book are excellent. Especially commendable, also, is the use of short paragraphs with topic headings, which breaks up the printed page and makes for easy reading. Occasionally the sentences are complicated, especially when precise definitions are attempted. References follow each chapter, and these are really suggestions for further reading, since usually the material of the references given is not referred to in the text.

Apart from these minor criticisms, the book has value, especially to those who need to be more familiar with the physiology of the binocular coordination of the two eyes as related to refraction procedure.

**Antibiotic Therapy.** By Henry Welch, Ph.D., and Charles N. Lewis, M.D. Price, $10. Pp. 562. The Arundel Press, Inc., P. O. Box 2606, Washington 13, D. C.

This book is a good reference volume for those interested in the properties and uses of the antibiotics, as well as their side-effects. Its field of usefulness to the ophthalmologist is limited, since there is almost no consideration of the use of antibiotics in ocular disease.

**Human Physiology.** By Bernardo A. Houssay, and others. Price, $14. Pp. 1118, with illustrations. McGraw-Hill Book Company, Inc., 330 W. 42d St., New York 18, 1951.

This recent addition to the growing number of texts on physiology will be particularly valuable to ophthalmologists, since the section on physiology of the eye is exceedingly well done and replete.

It can be recommended to everyone interested in a review of the recent developments of the physiology of the eye.

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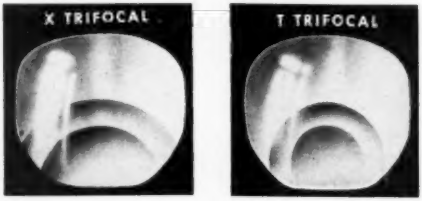


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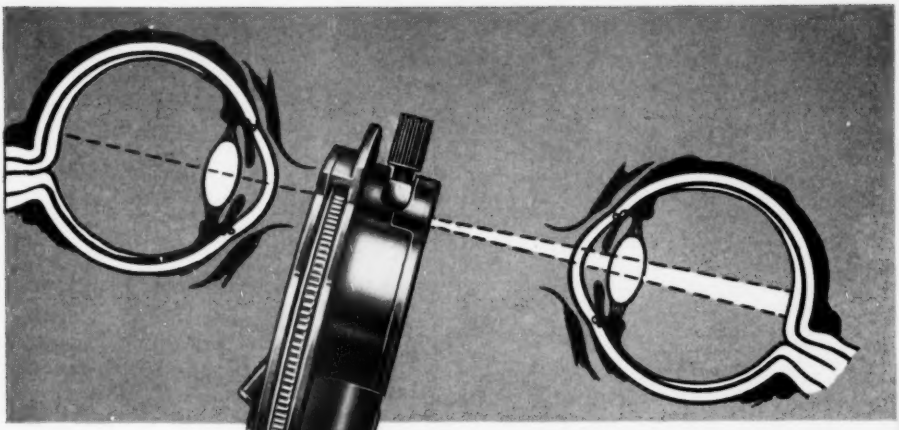

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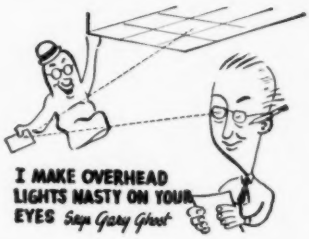


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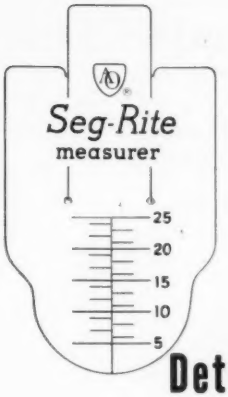


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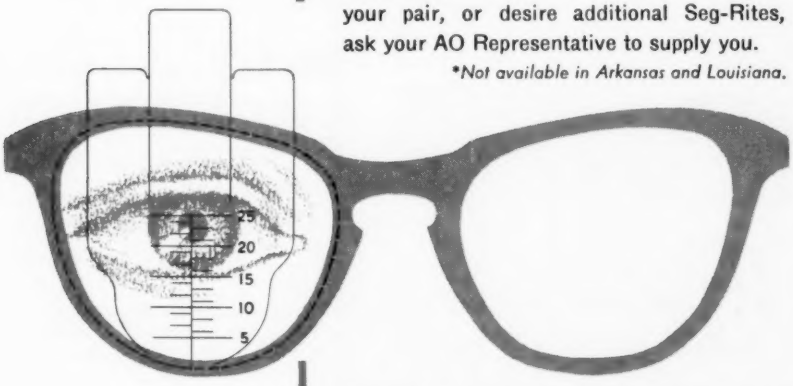


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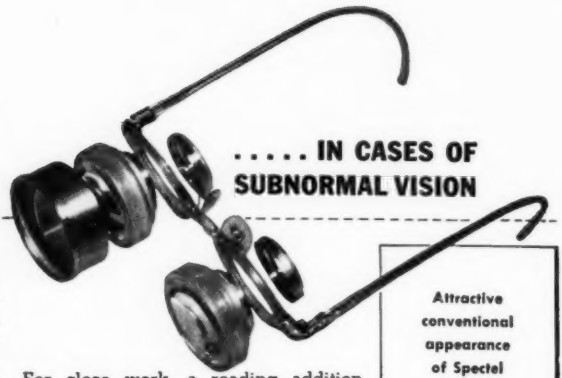

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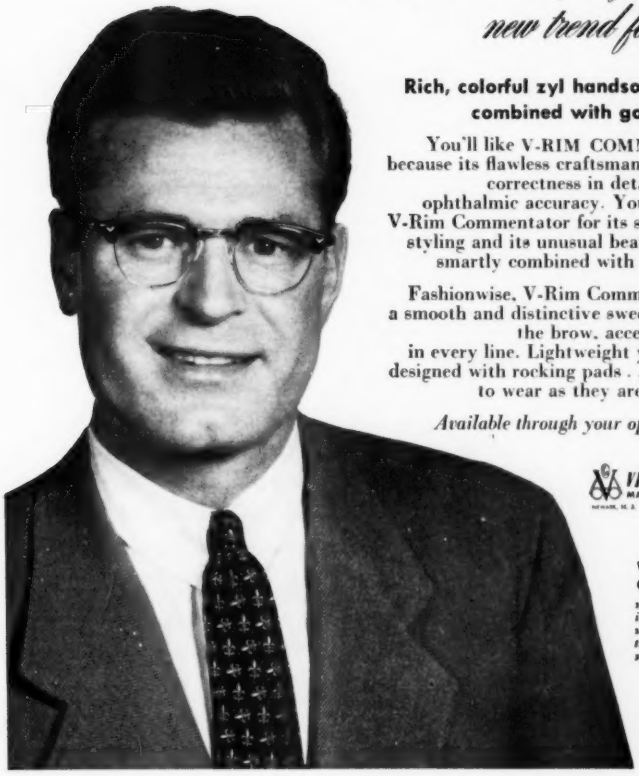

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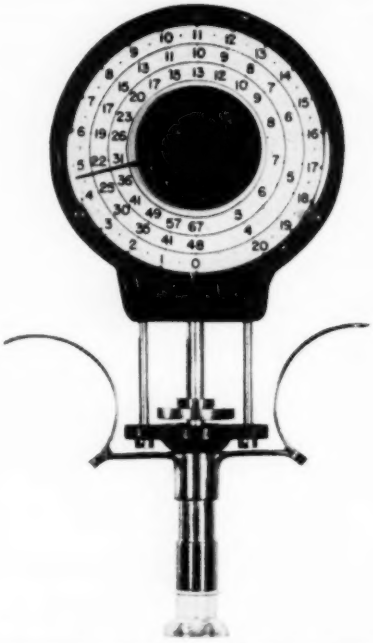

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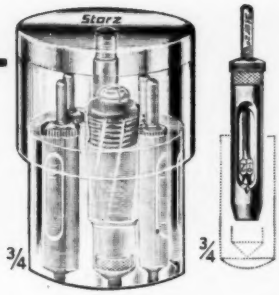


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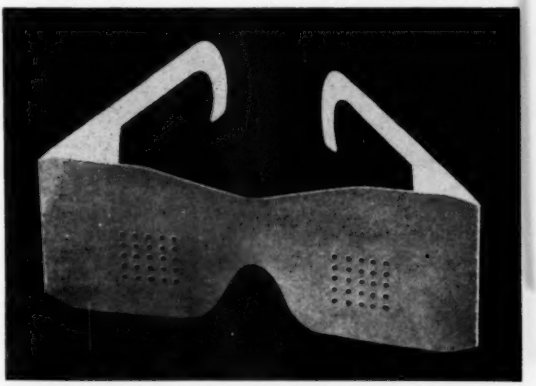

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